

Cardiology, DMCH – Questions & Answers (Ward Round)

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Q. Description of instrument –

TPM generator with 2 lead receiving port

TPM – a device that stimulate the heart temporarily with some preset parameters



Q. Temporary pacemaker (TPM) generator – Components

- **3 dials/ knobs (70/5/2)**
 - o **Rate** (30,40,50,60,70,**80**,90,100,110,120; orange coloured **140-160-180**) **60-70/min**
 - o **Output** in mA (0.1, 0.2, 0.5, 1, 2, **5**, 7, 10, 15, 20)
 - o **Sensitivity** in mV (orange coloured most **0.5**; 1, **2**, 3, 5, 10, 20 least, asynch.) (Ideally for ventricle: sensitivity 2)
- **2 buttons –**
 - o OFF (press both on & off button simultaneously for off)
 - o ON
- **3 indicators –**
 - o Pace
 - o Sense
 - o Low batt.
- **2 ends –**
 - o Proximal end – two lead receiving port
 - o Distal end – Battery cage (Alkaline 9.0 Volt) (PPM battery 2.8 V)
- Battery case
- **Modes: all TPM (VVI), AAI**

Q. TPM routes/ Routes of pacemaker lead implantation – (C M Shaheen Kabir p.48)

- **Trans-venous** – (mostly used)
 - Femoral vein (mostly used),(FV→ EIV→ CIV→ IVC→ RA→ RV apex)
 - Right IJV,
 - **Subclavian vein / Cephalic vein (left)** (PPM also)
 - basilic vein (Previously done) (Nazrul sir)
- Trans-cutaneous/ external (emergent situation- asystole, with high flow current, with 2 pads, anteriorly near V3 lead and posteriorly)
- Trans-oesophageal/ Trans-gastric
- Epicardial leads (Churchill's Pocketbooks Cardiology, 2e, p.370)
 - Infants – where transvenous leads might be 'outgrown'
 - AV block during cardiac surgery, especially aortic valve surgery
 - Recurrent infection of the endocardial system, IE,
 - Mechanical TV replacement

Subclavian route/ Jugular route:

Advantages – short route, technically easier, less chance of thrombosis

Disadvantages –

- Pneumothorax,
- Bleeding (non-compressible site),
- Brachial plexus injury

TPM is rate sensitive, amplitude sensitive.

Q. Highest HR settings in TPM – 180/min

Q. OUTPUT & THRESHOLD –

Threshold (Volt):

- Minimum voltage at which capture is achieved.
- The threshold should be <1.0 Volt for ventricular leads.

Output (mA): (SAFE PACING THRESHOLD)

- Minimum amount of current required to produce stable myocardial depolarization/ regular capture/ regular ventricular contraction. (Unit is mA)
- Safe multiple of threshold. ($O=2T+ 1$) is practiced in BD. Not universal rule.
Start with 5, gradually ↓; **THRESHOLD is slightly above the point of capture failure**

Threshold testing – (by generator manipulation)

- Start with RATE at least 10-20 bpm higher than intrinsic rate,
- Decrease the OUTPUT gradually, THRESHOLD is slightly above the point of capture failure (OUTPUT 5, 4, 3, 2, 1.8, 1.6, 1.4, 1.3, **1.0**, 0.9, 0.8, **0.5**, **0.2**, **0.1**)
- If just threshold is set→ not safety pacing, chance of **exit block**
(If patient become restless or due to movement of patient, inadvertently lead may be displaced or inflammatory oedema)
- **For ventricle, ideal threshold is 1 Volt** (may as low as 0.5)

Q. If output is 5, what is the threshold? (Gaffar sir)

- Threshold is not necessarily $T= (O-1)/2$, that is $(5-1)/2=2$

- Threshold is calculated by generator manipulation, Gradually decreasing 'Output' and observing 'Pace' & 'Sense' indicators. If at Output 1, Pace & Sense indicators blink alternatively, then Threshold is just above 1 (slightly above the point of capture failure). So, Output can be set safely at 3.

Q. SENSITIVITY & SENSING – (Churchill p.373)

Sensitivity: (mV)

- Minimum electrogram amplitude required to register as a sensed event. (Churchill) (Electrogram – intracardiac ECG, not electrocardiogram amplitude)
- Current (Voltage) required to detect a sensed event.
- **SENSITIVITY** is inversely proportional to **SENSING**.
Sensitivity $\propto$ (1/Sensing)
- Typical settings: 4V for ventricular sensing, 1.5V for atrial sensing. (Churchill)

Sensing: Capacity of a pacemaker to sense/ recognize intrinsic myocardial depolarization/ activity.

- If sensitivity is set too low, the device may respond to the T-wave or an extracardiac potential as a sensed event (Oversensing). (Churchill)
- If sensitivity is set too high, the device may not respond to a low-amplitude event, e.g. ectopic beat (Undersensing). (Churchill)

Oversensing → underpacing (undue inhibition)

Undersensing → overpacing

[R (pacing) on T (intrinsic) → ventricular tachyarrhythmias]

Numerically if sensitivity $\uparrow$, TPM undersensed

Numerically if sensitivity $\downarrow$, TPM oversensed

Sensing is inversely proportional to sensitivity. (Sensing $\propto$ 1/Sensitivity)

Q. Importance of sensitivity button in TPM generator – Demand pacing (VVI/AAI/DDD)

Pacemaker is given a capacity to SENSE.

1. To prevent oversensing (thus to prevent undue stoppage of TPM)
2. To prevent undersensing (thus to prevent R on T phenomenon & malignant arrhythmias, paced R on intrinsic T wave)
3. To conserve battery life

In **older TPM generator**, there was no sensitivity button → **Fixed rate pacing (VOO, AOO, DOO)**. (VOO: Ventricle paced, No sensing, No trigger/ inhibition).

Disadvantages of older TPM generator:

- Battery decay
- R on T phenomenon → VT, VF (paced R on intrinsic T wave)

Q. When to change sensitivity in pacemaker settings – (Gaffar sir)

Ideally for ventricle, sensitivity is 2 mV.

- Hyperkalaemia: Tall T waves
- Ebstein's anomaly: 'Himalayan' P waves

We have to increase sensitivity to undersense TPM, otherwise tall T waves/ Himalayan's p waves will be sensed as QRS and this OVERSENSING will lead to UNDERPACING

Q. Causes of pacing failure – (Gaffar sir)

- In case of RV pacing – infarction, fibrosis → High threshold
- If TPM lead is not placed in ideal place
- Displaced TPM lead
- Coronary sinus pacing (during TPM lead placement)
- Perforation of RV apex (pericardium), RAA (irritate right phrenic nerve)
- Patient on Amiodarone, Ic drugs (Flecainide)
- Inflammatory oedema at the site of TPM lead (RV apex) → Exit block

Q. TPM leads – Proximal end, distal end & shaft

- Proximal ends – 2 connectors for connection with lead receiving port of TPM pulse generator
- Distal ends – 2 electrodes, (-)ve cathode at the distal tip, (+)ve anode at the proximal ring
- Shaft – Insulation (silicon rubber) and 2 coiled conductors to prevent fracture

Difficulties during passage through TV –

- PVC (during lead advancement against closed TV), short runs of VT (Gaffar sir)
- Arrhythmias, bradyarrhythmia, asystole (Nazrul sir)

Post placement CXR – (C M Shaheen Kabir p.48)

- Must be evaluated for pneumothorax
- For **RV apex pacing**, in P/A view, the electrode tip should be located to the left of the spine, and directed **inferiorly and anteriorly**
- For **pacing through coronary sinus**, the electrode tip is to the left of the spine, and directed **posteriorly and superiorly (Upwards & backwards)** (Nazrul sir)



Q. TPM – lead direction in fluoroscopic guidance, A/P & Lateral view – for RV apex pacing

- **A/P view - Downward, forward & towards the diaphragm**
- Inadvertently lead can go to **Coronary sinus, RVOT**
- RV apex pacing –
 - ECG – wide complex, LBBB pattern
 - Low/ideal pacing threshold (1).
 - In case of high outout (10), causes diaphragm irritation (Rabbit kick) and patient feels discomfort
-
- Coronary sinus pacing –

- o ECG – narrow complex, inverted p in inferior leads
- o High pacing threshold
- o In case of high output (10), does not causes diaphragm irritation.

Q. TPM withdrawal/ weaning – 5 days protocol (C M Shaheen Kabir, p.50)

TPM can be kept for maximum 10-14 days.

At first, check the intrinsic (own) rate. When intrinsic rate is persistently >50/min with stable other haemodynamic parameters, try weaning

Day 1 – reduce rate by 10 for 1 hours (observe 'Pace' & 'Sense' indicators as well as monitor)

Day 2 – generator off on daytime only, at night it should be 'on'

Day 3 – generator off on day & night

Day 4 – generator off from connecting wire but leads in situ

Day 5 – leads off (preferably under fluoroscopic guidance)

Criteria for TPM withdrawal –

- Patient asymptomatic
- Haemodynamically stable
- Pulse, BP – within acceptable limit
- Sinus rhythm

Q. During TPM, drugs to stimulate native conducting system (SA node) –

- Propantheline (Tab. Prokind 15 mg, 1+1+1)
- Theophylline (Tab. Unicontin 200mg, 1+1+1)
- Amlodipine if hypertensive
- Nebulization with salbutamol ± ipratropium

Q. If patient on temporary pacemaker (TPM), feels dizzy – Possibilities –

- Oversensing → Underpacing (Undue inhibition)
- Pacing failure

Check the followings –

- Low battery indicator
 - Adjust rate (60-70/m)
 - Increase Output
 - Increase sensitivity (To prevent oversensing)
 - Any interruption of connection
 - If the lead is displaced, take the patient to cath lab for repositioning of lead
- (In short, INCREASE RATE, OUTPUT & SENSITIVITY)

Q. Patient on TPM, ECG/ monitor shows NO SPIKE – (Gaffar sir)

- Pacing failure
- Oversensing → Underpacing

Q. Patient on TPM, ECG/ monitor shows – (Gaffar sir)

SPIKE but No depolarization or

No association between spike & complex – Possibilities –

- Undersensing → Overpacing

- Capture failure

Q. Indications of TPM/ temporary pacemaker – (Nazrul sir) (Mir Jamal sir)

- Acute STEMI anterior/inferior MI with CHB
(Acquired CHB – symptomatic, rate <40/min, age – elderly, pause >3 seconds)
- Second-degree A-V block – if symptomatic
- Sick sinus syndrome (SSS) – documented bradycardia
- HCSS – recurrent syncope, asystole
- HOCM – if risk of SCD
- Congenital heart disease with CHB – ASD primum, TGA
- AF during ablation
- Bridging to permanent pacemaker (PPM), usually due to symptomatic degenerative/ congenital CHB
[Congenital CHB (Maternal SLE, Rubella) – symptomatic]
- Prophylactic TPM – Cardiac surgery including congenital heart disease, aortic surgery, high risk CAG) (during TAVI – keeping heart rate 180/min)
- Severe bradycardia (β -blockers, digoxin, verapamil, **hyperkalaemia**)

Q. Permanent Pacemaker – Indications of PPM (Wadud sir)

1. **Symptomatic degenerative CHB**
2. **Symptomatic congenital CHB**
3. Symptomatic second-degree heart block
4. **Post-MI –**
Persistent heart block (usually CHB) after 1 week (anterior MI) or **2 weeks (Inferior MI)**
(Wadud sir)
 - **CHB** or
 - Mobitz type II HB with BBB
 - New onset BBB with intermittent Mobitz type II HB
 - **Bifascicular & Trifascicular block** (Mohsin sir)**(Anterior MI with CHB, Inferior MI with persistent CHB)**
5. Syncope with sinus node dysfunction (**symptomatic SSS**)
6. Symptomatic chronotropic incompetence
7. Second or third degree heart block – associated with neuromuscular disease, after cardiac surgery
8. Chronic bifascicular & trifascicular block with intermittent CHB (Class I) (Wadud sir)
9. Symptomatic sinus bradycardia
10. Non-cardiac: HCSS, malignant VVS

Q. PPM generator –

(i.e. Dual chamber PPM generator with leads) DDDR, Medtronic

- Plastic part –
 - o Metallic cage (Titanium)
 - o Power source – Lithium Battery (**2.8 V**) (TPM battery – 9.0 Volts)
 - o Circuitry (Griffin 5e, p.771)
 - Output circuits
 - Sensing circuits
 - Timing circuits
 - Telemetry circuits

- Microprocessor
 - Sensor circuit for rate-adaptive pacing
- Metallic part –
 - Lead receiving port
 - Lead fixation screw
- Leads systems – Single chamber (1), dual chamber (2), CRT (3)
 - **Terminal pin** –
 - **Lead body** – [Conductor(s) + insulation]
 - **Stimulating/ sensing electrodes**
 - **Fixation device** –
 - **Passive fixation** – represents attachment mechanism ('fins' or 'tines')
 - **Active fixation leads** – secured to the endothelium using a 'screw-in' mechanism.
 -

Q. Lead fixation methods – (Churchill, p.369)

- **Endocardial leads** – implanted via the cephalic or subclavian vein; tines or fins allow the leads to lodge into trabeculae
- **Atrial leads** – J-shaped, designed to hook into the RA appendage (RAA)
- **Ventricular leads** – longer and straight, designed to lodge the RV apex. There are two principal methods for fixing these leads to myocardium:
 - **Active fixation leads** – use a screw tip to secure the lead into the atrial or ventricular wall. During insertion, the lead is screwed in over a stiff wire or stylet. Although initial pacing thresholds are usually higher than with endocardial leads, long term performance is good. **Steroid-eluting leads** have a steroid eluting tip which reduces inflammation after insertion, and improves pacing threshold and sensing.
 - Indications:**
 - Difficulty securing stable position with passive fixation lead,
 - Unexplained lead displacement,
 - Massively dilated right heart (RAA difficult to locate, RV apex and trabeculae less likely to 'hold' passive RV lead, TR displaces lead back into RA)
 - **Passive leads** – have either tines or fins at the tip, which allow the lead to snag in trabeculae. These are suitable for most patients and generally produce **low threshold** at implant.

Q. Pacemaker leads – (Churchill, p.369)

Lead Polarity:

- **Bipolar lead** –
 - Two terminals at the lead tip; current path is short (**small electrical field**) and the pacing signal often small (**small spike**) or invisible on the surface ECG.
 - Extracardiac potentials (e.g. from chest wall muscle) are less likely to interfere with function. Other than coronary sinus leads, used in CRT, most current pacing leads are bipolar.
 - Lead tip is negative (cathode),
 - Proximally positive (anode).
- **Unipolar leads** –
 - Have one terminal (–); the device can act as the +ve terminal.
 - Large spike

- Nowadays only LV CRT leads or epicardial leads are unipolar.

Bipolar lead	Unipolar lead
1. 2 electrodes are at the end of the lead	2 electrodes remain away from each other (cathode is on the lead tip & anode is the pacemaker can)
2. 2 conductors	1 conductor
3. Wide QRS	Narrow QRS
4. More chance of fracture	Less chance of fracture
5. Sensing area is small	Sensing area is large
6. Electromagnetic Interference (EMI) are less as the current path is short	Electromagnetic Interference (EMI) are more
7. Minor lead displacement can cause greater problem	No major problem
8. Pacing spike small may be invisible on the surface ECG	Pacing spike large on the ECG

(C M Shaheen Kabir, p.45)

In PPM, epicardially placed leads result in **smaller pacing spikes** than endocardially placed leads. (LITFL.com)

Q. PPM - Ventricular pacing – ECG morphology

- RV pacing lead placement → QRS morphology similar to LBBB
- Left epicardial pacing lead placement → QRS morphology similar to RBBB

Q. North American Society of Pacing & Electrophysiology (NASPE) & the British Pacing & Electrophysiology Group (BPEG) – Pacemaker codes

NASPE/BPEG Generic (NBG) Pacemaker Code – (5 codes):

NAPSE/BPEG generic pacemaker codes (Churchill)				
Chamber(s) paced	Chamber(s) sensed	Response to sensing	Rate response	Anti-tachycardia functions
0 – None	0 – None	0 – None	0 – None	0 – None
A – Atrium	A – Atrium	T – Triggered	R – Rate response	P – Anti-tachycardia pacing
V – Ventricle	V – Ventricle	I – Inhibited	M – Multiprogrammable*	S – Shock
D – Dual	D – Dual	D – Triggered and Inhibited		D – Pace & Shock

* used occasionally

Last revised in 2002 (previous version 1987) (Baltazar)

- Position I – Chamber (s) paced – O, A, V, D $D=(A+V)$
- Position II – Chamber (s) sensed – O, A, V, D $D= (A+V)$
- Position III – Response to sensing – O, T, I, D $D= (T+ I)$
- Position IV – Rate modulation – O, **R**
- Position V – Multisite pacing – O, A, V, D $D=(A+V)$
(Anti-tachycardia)

Fourth letter – identifies programmable features of pacemaker. (Baltazar)

- P – Programability is simple
- M – Multiprogrammable
- C – Communicating,
- R – Rate modulating.

Fifth letter – wheather the pacemaker is capable of terminating tachyarrhythmias (Baltazar)

- P – Pacing is used to terminate tachyarrhythmias,
- S – Shock is used to terminate tachyarrhythmias,
- D – Dual, both pacing & shock can terminate tachyarrhythmias.

Common pacing modes:

- AAI in sinus node dysfunction with intact AV conduction
- VVI in AF, atrial flutter
- DDD – commonest pacing mode
- Magnet mode

Pacing modes:

VOO:

- Paces at a fixed rate regardless of intrinsic rhythm.
- No longer used except to provide fixed rate pacing during surgery, to prevent inhibition by diathermy potentials.
- Theoretical risk of inducing ventricular arrhythmias by pacing during T wave (paced R-on-T phenomenon)

VVI:

- Cheap.
- Inhibited by sensed QRS events, so inappropriate pacing seen with VOO mode is prevented.
- **Indications:** AF + CHB, symptomatic bradycardia
- **Disadvantages:** AV synchrony is lost. (Pacemaker syndrome)

AAI:

- **Indications:** Sick sinus syndrome with intact AV node conduction.
AAIR for chronotropic incompetence.
- **Disadvantages:** AV block may develop later on in <2% of these patients per year.

DDD:

- Most physiologic pacing mode.
- The pacemaker can be
 - totally inhibited with normal sinus rhythm (As-Vs),
 - can pace the atrium with spontaneous ventricular depolarization (Ap-Vs),
 - can pace the ventricle in response to a spontaneous P-wave (As-Vp) and
 - can sequentially pace both the atrium and ventricle (Ap-Vp)
- **Indications:** AV block with intact sinus node function.
- DDIR for chronotropic incompetence.

- **Disadvantages:** Rapid ventricular pacing may occur during atrial tachyarrhythmias, which may necessitate device reprogramming. Mode switching devices partly circumvent this problem.

DDI:

- Similar to DDD mode, **tracking does not occur** in response to atrial activity.
- **Indications:** **rarely used** as pacemaker rate is fixed.
 - Sick sinus syndrome.
 - DDIR if (SSS+chronotropic incompetence)
 - Hypersensitive carotid sinus syndrome (with hysteresis)
 - Malignant vasovagal syncope (with hysteresis)
- **Disadvantages:** does not maintain AV synchrony during AV block.

VDD:

- Single ventricular lead but also have 'floating' sensing electrodes in the RA (atrial portion of the ventricular lead) to allow tracking of atrial rhythm.
- **Indications:** **(rarely used)**
High grade AV block with intact sinus node function.
- **Disadvantages:** Atrial undersensing in <5% of case, VVI pacing during sinus bradycardia

Q. DDDR – (Atahar sir) (C M Shaheen Kabir, p.56)

- 1st D – Chamber paced, atrium & Ventricle, **Dual pacing**
- 2nd D – Chamber sensed, atrium & Ventricle, **Dual sensing**
- 3rd D – Response to sensing, triggered or inhibited (**Mode of activation**) (Wadud sir)
- R – Rate responsiveness

Q. Sequential pacing & synchronous pacing – (Wadud sir) (Gaffar sir)

Sequential pacing:

- **Dual chamber pacing with (fixed) paced AV delay,**
 - maintaining A-V synchrony.
 - [Atrial paced (Ap) – paced AV delay – Vp (Ventricle paced)] (Ap-pAV-Vp)
 - DDDR
 - Not sinus rhythm
- (but if sensed atrial rhythm, sensed AV delay but paced ventricular rhythm – sinus)

Synchronous pacing:

- Maintaining A-V synchrony
- **DDDR (used in Sinus node dysfunction),**
- AAI (Ap-pAV-Vs) (VVI is not synchronous pacing)
(AAI is not sequential pacing)

All sequential pacing are synchronous pacing, but all synchronous pacing are not sequential.

Baltazar p.428. (DVI – sequential but not synchronus.)

Synchronized pacing: DDDR, AAI

Synchronized but not sequential: AAI

Q. Adaptive functions of pacemaker/ Adaptive pacing features – (Churchill, p.370)

- **Rate-responsive pacemakers:** It uses accelerometer, minute ventilation, temperature or QT interval sensors to trigger an increase in HR in response to activity. The degree to which, and rate at which HR increases is programmable.
- **Mode-switching pacemakers:** Used for patients with AV block and intermittent atrial tachyarrhythmias. Dual chamber pacemakers (in DDD mode) attempt to pace the ventricle rapidly in response to atrial rhythm during tachycardia. Mode-switching devices convert to VVI or VVIR mode if the atrial rate exceeds a programmed threshold. Almost all contemporary device carry a mode switch function.

Multiprogrammability: Ability to adjust device's pacing & sensing settings (e.g. rate, output, sensitivity) to suit the individual. All modern devices are multiprogrammable.

Q. Hysteresis – (Griffin 5e, p.779)

- A pacing feature where the **takeover rate of pacemaker is lower than the pacing rate**. A pacemaker with a pacing rate of 70/min and hysteresis mode set at 50/min will not start pacing until the patient's HR falls to <50/min, then the pacing rate jumps to 70/min. This function is useful in **transient bradycardias** occur in patients with following conditions to augment cardiac output during attacks while preserving sinus rhythm at other times.

- Carotid sinus syndrome (HCSS),
- Malignant Vasovagal syndrome.

(C M Shaheen Kabir, p.60)

Advantages:

- 1) It conserves battery life.
- 2) Maintains physiological intrinsic rhythm. (C M Shaheen Kabir, p.60)

Disadvantages:

- 1) Hysteresis can appear as a long delay on the ECG strip, it can be misinterpreted as failure to pace.
 - 2) Inappropriate tachycardia. (C M Shaheen Kabir, p.60)
- Pacing feature that attempts to allow the heart's native conduction system to predominate and therefore modifies base rate behavior.
 - This feature works by **using a longer escape interval after a sensed beat** than after a paced beat.
 - For example, the device sets the hysteresis rate at 50/min whereas the basal rate is 60 beats/min. Therefore, if the patient's intrinsic rate is >50/min, the device will not pace. However, if the patient's rate falls below 50/min, the pacer will pace at 60/min.
 - As hysteresis can appear as an unusually long delay on the ECG strip, it can be misinterpreted as **failure to pace** or as **inappropriate-sensing**.

Q. Escape interval and Pacing interval – (C M Shaheen Kabir, p.60)

Escape interval (V-A interval): The interval between an intrinsic beat and pacing beat.

Pacing interval: The interval between two consecutive pacing beats.

Q. Pacemaker malfunctions: LITFL.com

- **Problem with pacing –**
 - Output failure/ Failure to pace –
 - Failure to capture –
- **Failure to sense –**
 - Oversensing → Underpacing
 - Undersensing → Overpacing

- **Pacemaker associated dysrhythmias –**
 - Pacemaker-mediated tachycardia (PMT) – due to V-A conduction
 - Sensor-induced tachycardia – due to physiological stimuli (exercise, tachypnoea, hypercapnoea, acidaemia)
 - Runaway pacemaker – due to battery depletion
 - Pacemaker-mediated Wenckebach AV block
 - Lead displacement dysrhythmia

Q. Long-term complications of RV pacing – (Gaffar sir)

- Pacemaker dependence
- Gross-dyssynchrony
- Cardiomyopathy

His-bundle pacing (HBP): To avoid dyssynchrony (AV, inter- & intraventricular dyssynchrony).

Q. Patients with PPM presented with rate 115/min – possibilities are – (Gaffar sir)

- Sensor-mediated tachycardia (body temp, CO₂, Metabolic demand, muscular activity)
- PMT
- Tracking of atrial tachy-arrhythmias (Atrial tachycardia, AF) to ventricle
- ? Pacemaker-mediated Wenckebach
(If rate 300/m, Runaway pacemaker)

Q. Pacemaker-mediated tachycardia (PMT)/ Endless-loop tachycardia/

Pacemaker circus movement tachycardia: (Griffin 5e, p.787)

- **Re-entry tachycardia**, in which the pacemaker forms the antegrade pathway with retrograde conduction occurring via AV node
[Repetitive sequence of sensing of retrograde atrial activity results in triggering of a ventricular-paced beat at the end of the MTRI (maximum tracking rate interval)] (Griffin)
- **Triggers:** PVC, atrial undersensing, atrial oversensing, atrial non-capture (Griffin)
- Caused by retrograde p waves being sensed as native atrial activity with subsequent ventricular pacing
- The paced ventricular complex results in further retrograde conduction (V-A) with retrograde p wave generation, thus forming a continuous cycle
(ELT is sustained until there is VA block) (Griffin)
- Results in a paced tachycardia with the maximum rate limited by the pacemaker programming
- Can be **terminated by** slowing AV conduction e.g. adenosine or activation of **magnet mode**
- Newer pacemakers contain programmed algorithms designed to terminate PMT
[PVARP (Post-ventricular atrial refractory period) to a value that exceeds the VA conduction interval] (Griffin)
- May result in **rate-related ischaemia** in the presence of IHD

Q. Sensor-induced tachycardia –

- Modern pacemakers are programmed to allow increased heart rates in response to physiological stimuli such as exercise, tachypnoea, hypercapnoea or acidaemia
- Sensors may 'misfire' in the presence of distracting stimuli such as vibrations, loud noises, fever, limb movements, hyperventilation or electrocautery
- This misfiring leads to pacing at an inappropriately fast rate

- The ventricular rate cannot exceed the pacemaker's upper rate limit (usually 160-180/min)
- These will also usually terminate with application of magnet.

Q. Runaway pacemaker: (Gaffar sir)

- This potentially life-threatening malfunction of older-generation pacemakers is related to **low battery voltage** (e.g. overdue pacemaker replacement)
- The pacemaker delivers paroxysms of pacing spikes at 2000 bpm, which may provoke VF
- Paradoxically, there may be failure to capture – causing bradycardia – because the pacing spikes are very low in amplitude (due to the depleted battery voltage) and because at very high rates the ventricle may become refractory to stimulation.
- **Application of a magnet** can be life saving but definitive Rx requires replacement of the pacemaker

Q. Magnet mode of permanent pacemaker: (Gaffar sir) LITFL.com

- Applying a magnet to a pacemaker will initiate the magnet mode
- **Fixed asynchronous mode** (AOO, VOO or DOO)
- **Magnet mode nullify external interference & ignore intrinsic cardiac activity**
- Asynchronous modes deliver constant rate paced stimuli regardless of native rate of rhythm
- In asynchronous ventricular pacing, there is a risk of pacemaker-induced VT (The response of a pacemaker when a magnetic field of sufficient strength is applied and closes the pulse generator reed switch. The pulse generator paces at a predetermined rate & mode, which vary among pacemaker models & manufacturers.) (Griffin 5e, p793)
- **Indications:** (to assess pacemaker function & battery status) (Griffin 5e)
 - **To see battery life (EOL)**
 - **During operative procedure** (electrocautery)
 - During accidental mode switching due to external interference (passage near electrical station, airport scanner etc.)
 - PMT, sensor-induced tachycardia

Q. Investigations for pacemaker malfunction –

- ECG, real-time telemetry
- Serum electrolytes (↑K causes ↑threshold)
- Serum calcium, Serum Magnesium
- Application of magnet (VOO mode) Asynchronous mode
- Troponin I (to see concurrent MI, pacing threshold ↑)
- Chest X-ray P/A view – to see physical integrity of lead system

Q. Pacemaker syndrome – due to A-V dissociation (Gaffar sir)

- Single chamber **ventricular** pacing (not single chamber atrial pacing)
- Atrial contraction against closed TV and MV,
- Right heart:
Neck & facial engorgement, facial flushing, neck fullness, conjunctival congestion, transient (throbbing) headache
- Left heart (forward failure):
Fatigue, **Dizziness, lightheadedness, presyncope, syncope**
Hypotension (↓SBP >20 mmHg during change from native rhythm to paced rhythm)
LITFL.com

- Left heart (backward failure): Transient SOB (pulmonary oedema), cough

Q. Twiddler's syndrome – LITFL.com

- Patient manipulation of the pulse generator (accidentally or deliberately)
- The pacemaker rotates on its long axis, resulting in dislodgement of pacing leads
- Can result in diaphragmatic or brachial plexus pacing (e.g. arm twitching) depending on extent of lead migration
- Pacemaker is turned upside down within the pacemaker pocket, usually unintentionally. (Griffin)
The leads may become twisted, resulting in excessive traction on the leads and dislodgement

Q. Acute complications of pacemaker implantation – (Griffin 5e, p.784)

- Pneumothorax/ haemothorax
- Pacemaker pocket haematoma
- Cardiac or central venous perforation
- Diaphragmatic stimulation
 - Left diaphragm by RV apex lead at high pacing output.
 - Displaced atrial lead can stimulate right phrenic nerve & right diaphragm
- Local muscular stimulation
- Pacemaker malfunction
- Lead dislodgement/ damage, pacing lead fracture
 - Low impedance: Break in the insulation of the lead
 - High impedance:
 - Contained fracture of the lead conductor
 - Poor terminal pin to device connection
 - Lead dislodgement

Impedance (Z): Total resistance to the flow of current through a conductor.

Q. Chronic complications of pacemaker implantation – (Griffin 5e, p.785)

- Pacemaker system infection
- Intravascular thrombosis or obstruction (subclavian or axillary vein, SVC)
- Twiddler syndrome

Q. Death declaration of a patient with PPM/ TPM –

ECG – Only pacing spike, no myocardial depolarization

Normally, after pacing spike, it captures, then depolarization.

**Q. A patient with pacemaker ECG, spike only before QRS, no spike before p wave –
Is it single chamber (VVIR) or dual chamber (DDDR) pacemaker? (Gaffar sir)**

- Atrium sensed, sensed (& fixed) AV delay (fixed PR interval, maintaining AV synchrony), ventricle paced → (Dual chamber pacemaker)

Q. ECG – Large pacing spike before each QRS with A-V dissociation (Wadud sir)

- Single chamber (Ventricular) pacemaker: Variable PR interval
- Dual chamber pacemaker: fixed PR interval.
- Large spike → Unipolar lead (Circuit: Lead tip to generator case)
- Small spike → Bipolar lead

Q. Patient with CHB with moderate pericardial effusion and BHL. Dx – (Atahar sir)

- Sarcoidosis

Q. Classify PPM. (Gaffar sir)

- Single-chamber PPM (VVI, AAI)
- Dual-chamber PPM (DDDR)
- CRT

Q. Classify ICD. (Gaffar sir)

- Single-chamber ICD
- Dual-chamber ICD
- CRT-D (Triple chamber ICD)

Q. Indications of ICD (Implantable Cardioverter-Defibrillator) therapy – (Griffin 5e, p.362, 797)

Class I indications –

- 1) **Survivors of cardiac arrest**, because of VF or haemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible cause
- 2) Structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable
- 3) **Syncope of undetermined origin** with clinically relevant, hemodynamically significant sustained VT or VF induced during EP study
- 4) **LVEF $\leq 35\%$ because of prior MI, at least 40 days post-MI, and NYHA class II or III**
- 5) Nonischemic DCM, LVEF $\leq 35\%$, and NYHA class II or III
- 6) **LV dysfunction because of prior MI, at least 40 days post-MI, LVEF $\leq 30\%$, and NYHA class I**
- 7) NSVT because of **prior MI**, LVEF $\leq 40\%$, and inducible VF or sustained VT during EPS
- 8) Symptomatic sustained VT in association with congenital heart disease after hemodynamic and EP evaluation

Class I indications for ICD therapy post-MI: (Amal sir) (Griffin 5e, p.69)

- ICM, EF $< 35\%$ with NYHA class II or III symptoms **at least 40 days post-MI.**
- ICM, EF $< 30\%$ with NYHA class I symptoms at least 40 days post-MI.
- ICM, EF $< 40\%$ with inducible VF or sustained VT on EP study.

Patients who undergo either percutaneous or surgical revascularization after MI should have as assessment of LV function **after 3 months** to help to determine the appropriateness of ICD implantation.

Reduced LV function (EF $< 40\%$) – best predictor of mortality.

- Measurement of LV function soon after MI may reflect **myocardial stunning**.
- So echocardiography should be remeasured at the time of possible ICD implantation, **usually 40 days after MI** for primary prevention or **3 months** if reperfused after MI, via either PCI or CABG.

Class IIa –

- 1) Unexplained syncope, significant LV dysfunction, and nonischemic DCM
- 2) Sustained VT and normal or near-normal ventricular function
- 3) **HCM** with one or more major risk factors for SCD
- 4) **ARVD/C** with one or more risk factors for SCD
- 5) **LQTS** with syncope and/or VT while receiving β -blockers

- 6) Nonhospitalized patients awaiting heart transplantation
- 7) **Brugada syndrome** with syncope
- 8) Brugada syndrome with documented VT that has not resulted in cardiac arrest
- 9) **Catecholaminergic polymorphic VT** with syncope and/or documented sustained VT while receiving
- 10) Cardiac sarcoidosis, giant cell myocarditis, or Chagas disease
- 11) **Congenital heart disease** with recurrent syncope of undetermined origin in the presence of either ventricular arrhythmias during EP study

Class IIb –

- 1) Nonischemic heart disease, LVEF $\leq 35\%$, and NYHA class I
- 2) LQTS with risk factors for SCD
- 3) Syncope and advanced structural heart disease where thorough invasive and noninvasive investigations have failed to define a cause
- 4) Familial cardiomyopathy associated with sudden death
- 5) **LV noncompaction**
- 6) Recurrent syncope associated with complex congenital heart disease and advanced systemic ventricular dysfunction when through invasive and noninvasive investigations have failed to define a cause

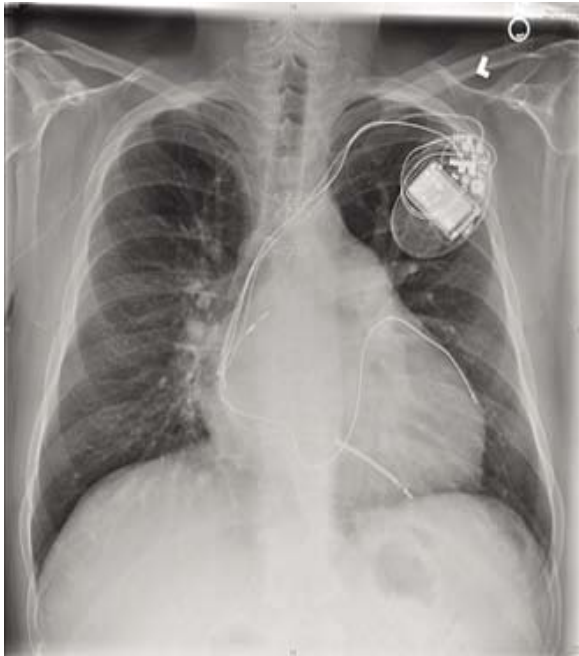
Q. 5 Criteria for CRT (Cardiac resynchronization therapy) – (Indications of CRT) (Wadud sir)

- Biventricular pacing
- **Heart failure symptoms** (NYHA Class II, III or ambulatory IV symptoms on GDMT) with **repeated hospitalization** despite optimal medical therapy for at least 3 months, expected life expectancy $>1\text{yr}$
- ECG –
 - o Sinus rhythm,
 - o LBBB + QRS duration $\geq 150\text{ ms}$ (Class I),
LBBB + QRS duration 120-149 ms (Class IIa)
 - o Non-LBBB pattern + QRS duration $\geq 150\text{ ms}$ (Class IIa),
Non-LBBB pattern + QRS duration 120-149 ms (Class IIb),
Only in NYHA class III & ambulatory IV
- Echo –
 - o **LVEF $\leq 35\%$** , (LVEF $<30\%$ in NYHA Class I, Class IIb recommendation)
 - o LVIDd $>55\text{mm}$ (Gaffar sir)
- NYHA Class I, LVEF $<30\%$, LBBB pattern, ICM (Class IIb)

Q. Any indication of CRT in presence of RBBB? (Lutfor sir) – Yes (Class IIa, IIb)

Q. Describe the X-ray with diagnosis.

Write down the 2 acute complications during procedure.



Q. HCM with CHB – ICD or PPM?

- ICD single chamber/ dual chamber) (Because ICD has both pacing and defibrillation capacity)
(Triple chamber ICD: CRT-D)

Q. LV noncompaction (LVNC) – (Griffin 5e, p.563)

- Relatively rare congenital abnormality of myocardium
- Trabeculated appearance of the LV cavity
- Spongy myocardium that results from arrest in endomyocardial morphogenesis
- It is seen in <1% of adults
- Can occur in isolation or in association with other congenital anomalies or with chromosomal abnormalities
- Over time, it is thought that noncompaction can proceed to DCM with severe systolic dysfunction
- Inherited mainly through autosomal dominant mode of transmission, but X-linked transmission is also seen.

Q. Dominance of coronary artery –

- Right dominant – 85%, PDA from RCA
- Left dominant – 8%, PDA from LCX
- Co-dominant – 7%
- Super-dominant – PDA supplying apex, LAD type II

Q. Left main – (Shahin sir)

- Normal length: 10-20 mm
- Short: <10 mm
- Long: >20 mm
- Diameter: 3-6 mm

Q. LAD Types/ Types of LAD – (Diameter 2-5 mm, mean 3.6mm)

- Type I – ends before apex

- **Type II – upto apex**
- Type III – wind round apex
- Type IV – upto inferior wall (wrap-around LAD)/ → antero-inferior MI upto crux (Shahin sir)
(The LAD supplies $\geq 25\%$ of inferior wall)

Q. LAD branches – diagonal, septal perforators

- Prox. LAD: before D1
- Mid LAD: D1 to D2
- Distal LAD: after D2

Q. LCX branches – (Diameter 1.5-5.5mm, mean 3mm)

- OM (Obtuse marginal branch)
 - Proximal LCX – upto OM1
 - Distal LCX – beyond OM1
- Circumflex atrial branch (Khaleq sir)
- PDA & PLV branch (if left dominant)

Q. Portions & branches of RCA – (Diameter 1.5-5.5mm, mean 3.2mm)

- Proximal RCA – upto the origin of RV branch
- Mid RCA – upto PDA
- Distal RCA – PDA (Posterior descending artery) & PLV (Posterior left ventricular branch)

Branches of RCA –

- 1) Conus,
- 2) SA nodal,
- 3) RV branch,
- 4) Acute marginal,
- 5) PDA &
- 6) PLV (Posterior left ventricular) branch

Q. VASCULAR ACCESS SHEATH with dilator & side arm with 3 way stopcock – (CORDIS SHEATH – trade name)

Parts –

- Sheath proper with side arm (it contains a one way valve, to prevents oozing of blood or tissue fluid and to prevent air entry)
- Dilator

How to check, valve is patent – occlude distal end with finger, give saline through side arm, look for any leakage through proximal end

Sizes of Vascular access sheath: (Khalequzzaman sir) (Internal dia for VAS, outer dia for catheter)

- 5 Fr (Internal Dia = 0.33x5mm) – Blue
- 6 Fr (Internal Dia = 0.33x6= 2mm)– Green
- 7 Fr – Orange (Not pink)
- 8 Fr – Ash colour

Less the dia, better. More the dia, more bleeding chance, more vascular trauma.

- Conventional CAG through radial route: 5 Fr (3-5 Fr), smaller upto 3 Fr
- Conventional CAG through femoral route: 6 Fr (6 Fr= 2mm= Internal diameter of sheath proper)
- PTCA of LM lesion or bifurcation lesion, ASD/VSD device closure: 7 Fr (for PCI, 6-7 Fr) (Mohsin sir)
- PTMC: 8 Fr for venous access (Larger dia 8-9 Fr needed), 5-6 Fr for arterial access
- Aortic graft/ TAVI 22 or 23 Fr) (Mohsin sir)
- If inadvertently (iatrogenic) more dilatation done by blade, then more dia needed
- Now newer technique in radial intervention – sheathless (NO VAS)

Length: 11 cm

- Long sheath is needed for tortuous iliac artery, upto 23 cm (upto infrarenal)

Uses of VASCULAR ACCESS SHEATH –

- **Air free, stable**, bloodless **vascular access**
- Flushing
- Easy manipulation of catheter
- Easy exchange of catheter & guide wire
- Medication (UFH)
- Pressure study

Indications:

1. All cardiac catheterization – CAG, PAG
2. All intervention procedures – PTCA, PTMC (Not for balloon insertion)
3. Pericardiocentesis
4. TPM (Khalequzzaman sir)
5. Autotransfusion in case of haemopericardium leading to tamponade (Flushing, medications, pressure study) (C M Shaheen Kabir)

Vascular access sites: Femoral, radial, subclavian, jugular, brachial, (axillary if all occluded), Aorta (paravertebral, not in practice)

Duration of applying manual pressure of the puncture site following CORDIS SHEATH removal –

- Arterial puncture: 15-20 min
- Venous puncture: 5-10 min

Q. Complication of vascular access – (C M Shaheen Kabir)

- Bleeding – major, minor
- Haematoma – local, retroperitoneal (RPH)
- A-V fistula
- Dissection, even perforation (Khalequzzaman sir)
- Pseudoaneurysm
- Thrombosis, cholesterol embolism
- Delayed - Infection

Q. 3 grave complications of vascular access sheath introduction – (Amal K Chaw sir)

- Retroperitoneal haematoma (RPH)

- Thigh haematoma
- Large pseudoaneurysm

Q. Difficult vascular access –

- Obese, female
- Elderly (thickened vessel wall)
- PAD
- Patient on anticoagulant

Q. How to understand that catheter is engaged (A/P view) – (Nazrul sir)

- Sudden jerking movement (rocking movement) of the catheter tip
 - Operator experience/ feeling
 - Fluoro – test dose dye push
 - Pressure changes
-
- LCA engagement in left anterior aortic sinus (Tall peaked T waves in inf leads, ↑HR)
 - RCA engagement in right posterior aortic sinus (T inversions in inf leads, ↓HR, conduction defects) (Nazrul sir)

Q. TIMI flow grade – the distal angiographic contrast runoff
(TIMI – Thrombolysis in MI)

TIMI 3 – Normal distal runoff

TIMI 2 – Good

TIMI 1 – Poor

TIMI 0 – Absence of distal runoff

Myocardial blush grade (MBG):

MBG 0 –

MBG 1 –

MBG 2 –

MBG 3 –

No-reflow phenomenon:

Q. Damping & Ventricularization –

- **Damping** – fall of SBP and DBP (Double fall), due to **complete** occlusion of coronary artery.
- **Ventricularization** – fall of DBP, due to **partial** occlusion of coronary artery.
It simulates that catheter in the LV.

Causes of Damping & Ventricularization –

- Ostial stenosis
- Ostial spasm
- Narrow vessel
- Over-engagement/ tight engagement
- Over-sized catheter

- Catheter tip impingement on vessel wall

Ways to avoid damping –

- GTN spray (works on spasm)
- Small size catheter
- Hit & run angiogram
- Quadri-plan or bi-plan camera (Image intensifier)

Q. LV diastolic pressure – 0-8 mmHg (5 mmHg) (Gaffar sir)

- Low LV diastolic pressure → **Coronary filling**

Q. First views of CAG – (Gaffar sir & Nazrul sir)

- RAO CAUDAL (to see the severity of disease) (Khaleq sir)

But in text, A/P CAUDAL view

Q. Angiographic views – Basic rule of thumbs

- RAO –
 - Spine is on the left,
 - Apex point to the right,
 - RCA resembles 'L'
 - Catheter in left
- LAO –
 - Spine is on the right,
 - Apex point to the left,
 - LAD is on the left
 - Catheter in right
 - RCA resembles 'C'
- LCX lies closest to the spine, nearer to catheter
- If diaphragm is seen – Cranial
- Cranial views is taken to see **Distal** part of coronary artery – Mid & distal LAD
- Caudal views is taken to see **Proximal** part of coronary artery – Left main, Prox LAD, LCX (Gaffar sir)

• Left coronary artery imaging:

- **RAO CAUDAL:** (First view in DMCH Cathlab) (Morton)
 - LMCA bifurcation,
 - Origin & course of LCX, OM
 - Prox LAD (beyond Proximal segment, overlapping diagonals), but apical segment of LAD seen clearly.
 - Ramus intermedius branch
- **LAO CAUDAL (Spider view):** (Morton)
 - LMCA (foreshortened),
 - LMCA bifurcation into LAD & LCX,
 - Proximal & mid LCX, origin of OM
 - Proximal LAD (foreshortened)
- **LAO CRANIAL:** (Morton)

- o LMCA (slightly foreshortened),
 - o LAD and its septal branches (coursing to left) & Diagonal branches (coursing to the right), separated clearly
 - o LCX & its marginal branches (foreshortened & overlapped)
- **RAO CRANIAL:** (Morton)
 - o Mid & distal LAD & origin of diagonals (upwards)
 - o Prox LAD & LCX overlapped
- **A/P CRANIAL:** Mid & distal LAD
- **A/P CAUDAL:** LMCA in its entire perpendicular length (Morton)
- **LATERAL:**
 - o **Mid & distal LAD** (Nazrul sir), LAD & LCX are well separated, diagonals overlapped
 - o Ramus intermedius
- **LAD** is seen: RAO **CRANIAL**, LAO **CRANIAL**, A/P **CRANIAL** (mid & distal)
- **LCX** is seen: RAO **CAUDAL**, LAO **CAUDAL**
- **Right coronary imaging:**
 - LAO CRANIAL:
 - o RCA origin,
 - o Mid RCA,
 - o PDA bifurcation (crux)
 - o PDA & PLV slightly foreshortened
 - RAO:
 - o Mid RCA,
 - o Length of PDA & PLV
 - AP CRANIAL:
 - o Best view to see PDA & PLV in right dominance,
 - o RCA origin
 - o Mid RCA foreshortened
 - LATERAL:
 - o RCA origin
 - o Mid RCA
 - o PDA & PLV foreshortened
- **LIMA:** (Morton)
 - o LAO
 - o AP, RAO
 - o Turning the patient's head to the right or left can sometimes assist in LIMA angiography

Q. 3-way PLASTIC MANIFOLD – Parts

- Proximal end – plastic hub, port for attachment of dye syringe
- Distal end – rotator which is connected to the catheter
- Shaft – **3 side ports with regulator** (Proximal to distal)
 - o D – Dye
 - o S – Saline flush circuit
 - o P – Pressure transducer
- Hold with 45° angle, repeated stroking to allow air to come up.

- During pushing of dye, we will keep some dye syringe at the end (to prevent air entry)
- Indications: CAG, PAG, PCI, Peripheral intervention (Not PTMC)
- Advantages:
 - Air-free uninterrupted closed arterial circuit
 - This converts single vascular access to multiple access
 - Helps to delivery of drugs (Heparin, nitrate), dye; also to infuse fluid
- Sterilization by Ethylene oxide (EO)
- Metallic manifold – not used now because
 - expensive, require sterilization, complex handling

Q. Sterilization of cathlab instruments – (Gaffar sir)

- 2% Glutaraldehyde (Cidex)
- Ethylene oxide

Q. How much heparin is used for CAG & PTCA – (C M Shaheen Kabir, p.06)

- 1 ml Heparin = 5000 U
 - Radial intervention:
 - Femoral intervention:
- Standard practice: 1ml = 5000 U after sheath insertion.
- 2000-3000 U in smaller patients.
- Additional heparin 50-70 U/Kg if PCI to minimize the risk of thromboembolism.

Target ACT (activated clotting time) during PCI – 200-300 seconds

- Heparin dosing is closely monitored by an ACT (Roughly 300 sec is expected) (C M Shaheen Kabir, p.06)
- During CAG <200s
- During sheath removal <150s

Q. Complications of cardiac catheterization/ Complications of CAG – (Nazrul sir)

- Arrhythmia (VT, VF) (0.3%), cardiac arrest, MI (0.05%), acute LVF,
- Thrombo-embolic complications- stroke (0.05%), limb ischaemia
- Death (0.1%)

During puncture & VAS introduction:

- Hemorrhage (Haematoma), Vasospasm, VVS
- High puncture – retroperitoneal haematoma (RPH)
- Low puncture – Injury to profunda femoris, Pseudoaneurysm, Aneurysm, A-V fistula
- Femoral nerve injury

During catheter introduction:

- Catheter knot, catheter fracture
- Aortic dissection

During engagement and wire crossing:

- Damping, ventricularization
- Coronary artery dissection, coronary artery spasm

Contrast-related:

- Muco-cutaneous – itching, rash
- Bronchospasm,
- Anaphylaxis
- AKI, CIN (0.3%)

Q. Contrast-induced nephropathy (CIN) – An ↑ in serum creatinine by

- either ≥ 0.5 mg/dL or
- by $\geq 25\%$ from baseline value after exposure to a contrast medium.

Q. Contrast/ dye used in cath lab – (F Rahman)

- Maximum amount that can be used = $eGFR \times 3$
- If LV dysfunction –
 - Mild to moderate (EF 35-55%) – 1 ml/Kg
 - Severe (EF $< 35\%$) – 0.5 ml/Kg
- For CAG, average amount of dye needed = 50 ml
- Normal saline can be used pre, per and post-operatively to reduce risk of CIN
- $NaHCO_3$, NAC with fruit juice
- i.e. Iopamidol 100 ml (Lopidam 370 - Beximco) - Low osmolar Non-ionic
Iodixanol (Visipaque) – Iso Osmolar Non-ionic
- **Complications:**
 - Muco-cutaneous: Itching, rash,
 - Anaphylaxis
 - AKI, CIN

Q. Contraindications of CAG – (Wadud sir) (Anaemia aggravate angina)

- Do not CAG if Hb < 9 gm/dl
- Do not PCI if Hb < 10 gm/dl

Q. Left heart catheters – (C M Shaheen Kabir, p.14, 35)

JAMP

For coronary angiogram (CAG) –

- **Judkin's catheter left (JL 3.5 to 6)** and **Judkin's catheter right (JL 3.5 to 6)**
(Diagnostic CAG catheter Judkins type – left and right) (3.5, 4.0, 5.0, 6.0 cm)
(0.038 inch dia)
- Amplatz catheter left (AL) and right (AR), I to III
- Multi-purpose catheter (A1, A2, B1 and B2) (Both end and side hole)
- Pig tail catheter (0.038 inch dia, both end and side hole)
- Coronary bypass catheter (LCB or RCB)
- Internal mammary catheter (IM)

For PTCA –

- PTCA guiding catheter – right and left
 - Judkins
 - EBU/ Extra back-up (XB)
 - Amplatz

For LV graphy –

- Pigtail catheter

Q. JUDKINS CATHETER – (C M Shaheen Kabir, p.15)

- Length – 100 cm
- Diameter – 0.038 inch
- **Length of second arm** determines the **size of the catheter** (i.e. 3.5, 4.0, 5.0, 6.0 cm).
- **A 4 cm Judkins catheter** is appropriate **for most adult** patients for catheterization of the LCA & RCA.
- Size of the catheter depends upon – body surface area, weight, age (larger in elderly), aortic root dilatation seen in echo (Mohsin sir)

Uses of JL (Judkins left) – Diagnostic angiogram of LCA.

Uses of JR (Judkins right) –

- Diagnostic angiogram of RCA
- LIMA & RIMA visualization
- Selective renal angiogram
- Selective peripheral angiogram (PAG)

Q. Differences between Judkins guiding catheter and diagnostic catheter –

Judkins guiding catheter	Judkins diagnostic catheter
Uniform diameter throughout the length	Distal end is 2 Fr narrower than proximal end to prevent total occlusion
Soft tip for prolonged attachment	Tip is not soft
Stiffer, hard for extra back up support	It is not hard
Lumen is larger	Lumen is smaller
Shaft is more thicker & supported	Shaft is not thick & not supported
White rubber band (Mohsin sir)	
6 Fr – Green	
7 Fr – Orange	

What will be instruction to S/N – Suppose, Judkins Left, 6Fr, 3.5 size (Mohsin sir)

Q. Pigtail catheter – (C M Shaheen Kabir)

- Length – 110 cm, diameter – 0.038 inch
- Parts –
 - Proximal end – contain plastic hub for attachment of syringe
 - Distal end – smooth & tapered, pigtail shaped curvature; contains **both end and side holes** (5-12)
 - Shaft –

Uses of pigtail catheter – (Gaffar sir)

1. **LV graphy** (Other catheters – **Multipurpose catheter** with 1 end hole & 2 side holes, Halo catheter, Sones catheter)
2. **Root aortography –**
 - Anomalous coronary artery
 - **AR gradient,**
 - **Aortic aneurysm,**
 - Supravalvular AS,

- RSOV,
 - Non-selective CAG, or
 - Bypass graft arteriography
 - **Sideness of aortic arch** (Gaffar sir)
3. **Arch aortography –**
- PDA,
 - AP window,
 - CoA
 - Takayasu disease
 - Atherosclerotic narrowing of major branches
4. Descending thoracic aortography –
- CoA,
 - MAPCA
5. Visualization of LIMA & RIMA
6. Non-selective renal angiography
7. Non-selective peripheral angiography
8. During PTMC –
- Anatomical landmark,
 - Pressure study (transmitral PG),
 - Severity of MR
9. Pericardiocentesis

Indications of LV graphy – (Gaffar sir)

1. Qualitative assessment of ejection fraction (**EF**) – (visual estimation)
2. Identify regional wall motion abnormality (**RWMA**)
3. Identification of no. and site of **VSD** (60° LAO CRANIAL view)
4. Identification of LV aneurysm
5. Severity of valvular regurgitation (**MR view - RAO 30°**) (Gaffar sir)
6. LV end-diastolic pressure (Pressure study)

Ventriculography views –

- 30° RAO
- 60° LAO

Contraindications of LV graphy – (Gaffar sir)

1. **Severe LV systolic dysfunction** (low volume, non-ionic dye can be used)
2. **LVEDP > 25 mmHg** (If the decision is to proceed with LV graphy is made, S/L nitroglycerine should first be given to lower the LVEDP & use low osmolar contrast agent)
3. **LV thrombus**
4. Persistent **LV arrhythmia**
5. Significant left main disease
6. Prosthetic heart valve
7. Critical AS
8. Renal failure

Complications of contrast LV graphy –

1. Cardiac arrhythmias – PVC, VT, VF (VT & VF – immediate xardioversion)
2. Myocardial contrast staining
3. Embolism from thrombi or air

4. Fascicular block
5. Contrast related complications – Allergy, transient hypotension, vasomotor collapse

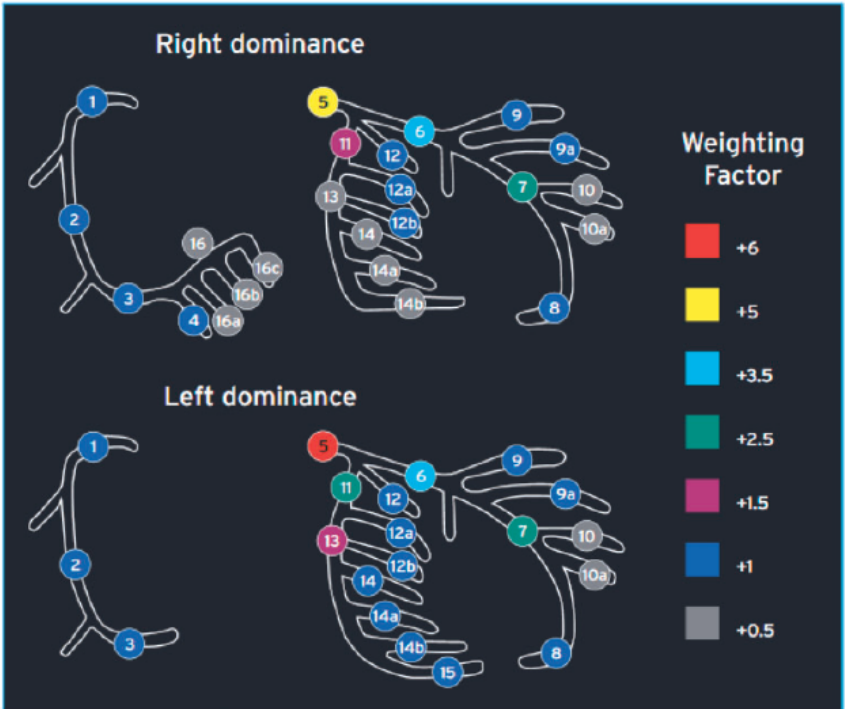
Q. Coronary angiogram (CAG) – Score system

1. TIMI flow:
2. Type of lesion: Type A, B and C
3. Vessel score: 1,2,3
4. Gensini score
5. Leaman score
6. Friesinger score/ index
7. Syntax score:
8. Coronary artery calcium (CAC) score –

Syntax score algorithm –

1. Dominance
2. Number of lesions
3. Segments involved per lesion, with lesion characteristics
4. Total occlusions with subtotal occlusions
 - I. Number of segments
 - II. Age of total occlusions
 - III. Blunt stumps
 - IV. Bridging collaterals
 - V. First segment beyond occlusion visible by antegrade or retrograde filling
 - VI. Side branch involvement
5. Trifurcation, number of segment diseased
6. Bifurcation type and angulation
7. Aorto-ostial lesion
8. Severe tortuosity
9. Lesion length
10. Heavy calcification
11. Thrombus
12. Diffuse disease, with number of segments

SYNTAX (**Sy**nergy between Percutaneous Coronary Intervention with **TAXUS** and Coronary Surgery): A largest prospective, randomized multicentre trial including ‘all-comers’ with complex MVD (left mainstem or complex TVD).

Steps	Variable assessed	Description
Step 1	Dominance	The weight of individual coronary segments varies according to coronary artery dominance (right or left). Co-dominance does not exist as an option in the SYNTAX score.
Step 2	Coronary segment	The diseased coronary segment directly affects the score as each coronary segment is assigned a weight depending on its location, ranging from 0.5 (i.e. the posterolateral branch) to 6 (i.e. left main in case of left dominance).  © ESC 2018
Step 3	Diameter stenosis	The score of each diseased coronary segment is multiplied by two in case of a stenosis 50–99% and by five in case of total occlusion. In case of total occlusion, additional points will be added as follows: ● Age >3 months or unknown +1 ● Blunt stump +1 ● Bridging +1 ● First segment visible distally +1 per non-visible segment ● Side branch at the occlusion +1 if <1.5 mm diameter +1 if both <1.5 mm and ≥1.5 mm diameter +0 if ≥1.5 mm diameter (i.e. bifurcation lesion)
Step 4	Trifurcation lesion	The presence of a trifurcation lesion adds additional points based on the number of diseased segments: ● 1 segment +3 ● 2 segments +4 ● 3 segments +5 ● 4 segments +6

Step 5	Bifurcation lesion	The presence of a bifurcation lesion adds additional points based on the type of bifurcation according to the Medina classification: ¹²⁶ - Medina 1,0,0–0,1,0–1,1,0 +1 - Medina 1,1,1–0,0,1–1,0,1–0,1,1 +2 Moreover, the presence of a bifurcation angle <70° adds one additional point
Step 6	Aorto-ostial lesion	The presence of aorto-ostial lesion segments adds one additional point
Step 7	Severe tortuosity	The presence of severe tortuosity proximal of the diseased segment adds two additional points
Step 8	Lesion length	Lesion length >20 mm adds one additional point
Step 9	Calcification	The presence of heavy calcification adds two additional points
Step 10	Thrombus	The presence of thrombus adds one additional point
Step 11	Diffuse disease/ small vessels	The presence of diffusely diseased and narrowed segments distal to the lesion (i.e. when at least 75% of the length of the segment distal to the lesion has a vessel diameter <2 mm) adds one point per segment number

Friesinger index: A score ranging from 0-15. Each of the three main arteries is scored separately receiving a score from 0-5.

Lesion grade –

- 0 – No disease
- 1 – Lesions <50% area stenosis
- 2 – Single lesion >50% but <90%
- 3 – Multiple lesion >50% but <90%
- 4 – 90%
- 5 – 100%

Q. Characteristics of ACC/AHA type A, B and C lesions –

Type A lesions (High success >85%, low risk)

Discrete (<10 mm length)	Little or no calcification
Concentric	Less than totally occlusive
Readily accessible	Non-ostial in location
Non-angulated segment <45 degrees	No major branch involvement
Smooth contour	Absence of thrombus

Calcification seen as **black lesion** (in contrast to CXR) in fluoroscopy (Khaleq sir)

Type B lesions (Moderate success 60-85%, moderate risk)

Tubular (10-20 mm length)	Moderate to heavy calcification
Eccentric	Ostial in location
Moderate tortuosity of proximal segment	Bifurcation lesions requiring double guidewires
Moderately angulated, 45-90 degrees	Some thrombus present
Irregular contour	Total occlusion < 3 months old

Type C lesions (Low success < 60%, high risk)

Diffuse (>2 cm length)	Degenerated vein grafts with friable lesions
Excessive tortuosity of proximal segment	
Extremely angulated, >90 degrees	Total occlusion > 3 months old (CTO)
Inability to protect major side branch	

Q. Chronic total occlusion (CTO) – (C M Shaheen Kabir, p.22)

- Duration of lesion – at least 3 months (based on H/O angina/MI)

- TIMI grade 0 or 1 flow

Q. Ostial lesion – Origin of the lesion <3mm of vessel origin. (~~Osteal~~) (Khaleq sir)

- More fibrotic
- Resistant to dilatation
- Supplies large area
- More risk of restenosis

Branch-ostial: LAD

Aorto-ostial: RCA

Q. Coronary bifurcation lesion –

- A lesion occurring **at, or adjacent to, a significant division** of a major epicardial coronary artery.
- **A significant side branch** is a side branch that the operator does not want to lose... a vessel that has a diameter >2.5 mm and/ or supplying large part of the myocardium
- **Medina classification** – [MB (proximal), MB (distal), SB];
Main branch (MB), Side branch (SB)
 - Each segment is assigned a value '0' in the absence of significant stenosis and '1' in the presence of disease (>50%).
 - A value of 0 or 1 is therefore assigned to each of the three segments separated in the following order, with number separated by commas.
 - o Proximal main branch,
 - o Distal main branch and
 - o Side branch.
 - 1,1,1 – Critical stenosis in all three segments
 - 1,1,0 – Only proximal & distal main branch affected
 - 1,1,0; 1,0,1; 0,1,1;
 - 1,0,0; 0,1,0; 0,0,1
 - Limitation: Length of the stenosis involving SB is not specified. (F Rahman, p.140)
- A coronary artery narrowing adjacent to and/or involving the origin of a side branch.
- Bifurcation lesion involves –
 - o Proximal main vessel
 - o Distal main vessel
 - o Side branch ostium

Q. Trifurcation lesion –

Q. Stents –

- **Bare metal stents**
- **Drug-eluting stents (DES)**
 - o **First generation DES** – Cypher (Sirolimus), Taxus (Paclitaxel)
 - o **Second generation DES with durable polymer coating –**
 - Everolimus coating: **Promus**, Promus Element Plus, **Xience** prime, Xience Xpedition, Synergy (Everolimus)
 - Paclitaxel coating: Joctax
 - Zotarolimus coating: Resolute Onyx, Resolute Integrity, Endeavor

- Biolimus coating: Nobori, Biomatrix
- Sirolimus coating: YukonChoice PC, Alex, Orsiro
- **Third generation stent:** Bioabsorbable vascular scaffold (BVS)
- **Newer DES:**
 - Biodegradable polymer coatings DES – Synergy (Everolimus), Ultimaster (Sirolimus), Combo (Sirolimus)
 - Polymer-free DES: CRE 8 (Amplimus)
 - Bifurcation stents
(Drug-eluting balloons -)
- On the basis of coating –
 - Bare metal stent:
 - Passive: Cobalt chromium, platinum chromium
 - Bioactive: Sirolimus, Paclitaxel, Zanolimus
- On the basis of composition –
 - Metallic
 - Polymeric
- On the basis of configuration –
 - Slotted tube
 - Coiled wire
 - Modular stents – Open cell design, highly flexible
 - Multicellular – Closed cell design, less flexible
 - Modular multicellular (hybrid stents) – combined effect
- On the basis of bioabsorption –
 - Inert/ biostable
 - Degradable/ bioabsorbable
- On the basis of mode of implantation –
 - Self-expanding
 - Balloon expandable
- On the basis of design –
 - Open cell design – more flexible, side-branch access, reduce radial support
 - Closed cell design – more support, less flexible
- Absorbable stents – Completely biodegradable, bioabsorbable stent, typically polymers or magnesium, sometimes coated with anti-stenotic agents e.g. Absorbable metal stent (AMS)
- Radioactive stents – stents with radiation emitting coating

Q. Nominal pressure & Rated burst pressure (RBP) –

Nominal pressure (NOM) – The pressure (ATM 8-22 or kPa) at which the balloon reaches its labeled diameter.

e.g Xience Prime 3.0 33mm NOM 10 atm pr. (3.01 mm), RBP 18 atm pr. (3.37mm)

RBP – The maximum pressure to which a balloon is designed to be inflated below which 99.9% of balloons will not rupture. The probability of balloon rupture is < 0.1%.

Q. Dye dilution for PTCA & PTMC –

- 1:1 dilution (1ml dye: 1 ml NS) for PTCA – Increase strength of dye is needed for better visualization.
- 1:4 dilution (1ml dye: 4 ml NS) for PTMC –

- for rapid inflation and deflation and
- to decrease viscosity

**Q. PTMC (Percutaneous Transvenous Mitral Commissurotomy)/
PMBV (Percutaneous Mitral Balloon Valvotomy) –
Indications, contraindications and procedure –**

Indications of PTMC: (C M Shaheen Kabir, p65) (AHA/ACC 2014)

- Symptomatic patients (NYHA functional class II, III or IV) with – **(Class I)**
 - **Moderate to severe MS**
 - **Favourable valve morphology for PMV (Percutaneous mitral valvotomy)**
 - **Absence of LA thrombus or moderate to severe MR**
- Asymptomatic patients with – **(Class I)**
 - **Moderate to severe MS**
 - **Favourable valve morphology for PMV**
 - Pulmonary hypertension
(PASP >50mmHg at rest or >60mmHg with exercise)
 - **Absence of LA thrombus or moderate to severe MR**
- Moderate to severe MS with a **nonpliable, calcified valve**, NYHA III-IV, **either not candidate for surgery or at high risk of surgery** **(Class IIa)**

Contraindications of PTMC:

- LA or appendage thrombus
- Moderate to severe MR
- Severe TR
- Severe pulmonary hypertension
- Wilkins score >8 (commissural fusion in PSAX) (Wadud sir)
- Patients with MS and aortic lesion that require cardiac surgery
(If CABG is needed for CAD, simultaneously OMC can be done.) (Wadud sir)

Echo score assessment for PTMC (Wilkins score)

- **Mobility** (Grade 0-4)
 1. Highly mobile with **only leaflet tips** restricted
 2. **Mild leaflet restriction**; base portions have normal mobility
 3. **Valve moves forward in diastole, mainly from base**
 4. No or minimal diastolic movement of valve
- **Subvalvular thickening** (Grade 0-4)
 1. **Minimal thickening below leaflets**
 2. Chordal thickening up to **one-third** of chordal length
 3. Thickening extending to **distal one-third** of chorda
 4. Extensive thickening of papillary muscles
- **Leaflet thickening** (Grade 0-4)
 1. Near normal (4-5 mm)
 2. **Marginal thickening** (5-8mm) with normal thickness of midleaflets
 3. **Thickening of entire leaflet** (5-8mm)
 4. Extensive thickening of all leaflet tissues (>8-10mm)
- **Calcification** (Grade 0-4)
 1. **Single area of echo brightness**

2. **Scattered areas** of increased brightness along leaflet margins
 3. Brightness **extending to the midportion** of the leaflets
 4. **Extensive brightness** throughout the leaflet tissue
- **A total echocardiographic score** (adding the four components) **higher than 11** is associated with poor outcome & suboptimal increase in MVA.
 - Score **9-11** represent gray zone, some have good results, some have suboptimal results
 - Optimal results of PTMC are usually achieved – when score is **8 or less**.

INOUE BALLOON CATHETER: Parts – Proximal end, distal end, and shaft

- Proximal end – has a **W-connector** consists of long side vent, central vent and short side vent with an inner metallic tube.
- **Long side vent** with 3 way stop cock is used to free the balloon from air
- **Short side vent** is used to inflate/ deflate balloon.
- **Central vent** is used to introduce Stretcher and Shepherd.

INOUE TECHNIQUE:

1. Antibiotic prophylaxis
2. Introduce **both arterial and venous sheath** through right femoral access
3. **Pigtail catheter** is advanced through arterial sheath and positioned in noncoronary aortic sinus (**Anatomical landmark, BP monitoring**)
4. Introduction of **Mullin's sheath** through venous sheath under guidance of terumo guide wire and is placed into **left brachiocephalic vein**
5. Terumo wire is removed and **Brockenbrough needle** is advanced inside the Mullin's sheath and is kept 1 cm inside the Mullin's sheath to avoid injury.
6. Then pull down the **Mullin's sheath including the needle** upto fossa ovalis (FO) of IAS under fluoroscopy guidance. There will be jerky feelings.
7. Distal tip of Brockenbrough needle is being advanced through Mullin's sheath for IAS puncture.
8. IAS is punctured with Brockenbrough needle (**Transeptal puncture**)
9. Brockenbrough needle is removed and **septogram** is done.
After confirmation of septal puncture **IV 1000 Units UFH** is given.
(Q. of Shafi Mozumder sir) Ideally UFH – should be given after septal puncture as LA is a prothrombotic environment.
10. **Spring tipped guide wire** is advanced through the Mullin's sheath & 2 and half circle is kept in the LA.
11. Then the Mullin's sheath including the venous access is removed.
12. Groin is dilated with dilator & then advanced over the spring tipped guide wire to dilate the puncture site of IAS.
13. Then dilator is removed.
14. Prepare the balloon.
15. Selection of balloon size is base on patient height.
16. Balloon size (mm) for upto 150 cm height = Patient height (cm) / 10 + 10
17. The **INOUE BALLOON CATHETER with BALLOON STRETCHER** is advanced through the IAS over the spring wire.
18. After that spring guide wire and balloon stretcher is removed.
19. **Shepherd wire** is advanced within the balloon **for positioning of the balloon in mitral valve**.
20. Then Shepherd wire is removed and **INOUE balloon inflation** is performed at first distal and proximal then middle part of commissural dilatation.

21. After dilatation **IV UFH 5000 Units** is given.
22. The balloon catheter is withdrawn into LA and transmitral gradient is immediately assessed.
23. Then coiled spring guidewire is introduced again along with balloon stretcher inside the INOUE balloon.
24. Balloon is removed with stretcher.
25. Spring guide wire is removed.
26. Then **LV graphy** is done by pigtail catheter to see mitral regurgitation
27. Arterial access is removed & proper haemostasis is ensured.

Complications of PTMC: Immediate complications:

CARDIAC:

- **Cardiac perforation** (2-4%)
- **Haemopericardium**
- Arrhythmia
- Cardiac arrest
- **MR** (3-8%), **Residual ASD**, MI
- Thromboembolism (2%)
- IE

LOCAL:

- Bleeding, Haematoma,
- Thromboembolism,
- AV fistula,
- Infection

Q. Criteria for successful PTMC – (CM Shaheen Kabir, p.68)

- Symptomatic relief
- Decrease in intensity and duration of MDM
- Complete opening of at least one commissure
- Increase in MVA by $> 1.0 \text{ cm}^2/\text{m}^2$ body surface area or by **at least 50%** (Wadud sir, not 80%), mainly as a result of splitting the fused commissure
- An immediate reduction of LA pressure to $<18 \text{ mmHg}$
- 50-60% reduction in trans-mitral gradient
- No systolic murmur on auscultation

Q. Before PTMC, is CAG is necessary – (Q of Shafi Mozumder sir)

- For assessment of high-risk patient – concomitant CAD, LV dysfunction
- When MV is occluded balloon, $\uparrow$ chance of arrhythmia in patients with CAD

Q. PTMC – (Gaffar sir)

- During PTMC, chance of pericardial tamponade during IAS puncture due to inadvertently free RA wall puncture
- Hugely dilated LA causing bulge of IAS towards RA – contraindication of PTMC

Pericardial tamponade complicating PTMC –

- S/S – Lightheadedness, heaviness of chest, palpitation, dizzy spells
- Fluoroscopic monitor – Heart movement almost absent, $\uparrow$ Cardiac silhouette

- Haemodynamic monitor – ↑HR, ↓SBP
- Dye – Heart floating on dye
- Auto-transfusion

Q. Management of Mitral stenosis – (Khaleq sir)

- Intervention: PTMC (Surgical counterpart - CMC)
- Surgical:
 - o CMC (Closed mitral commissurotomy) – No cardiopulmonary bypass needed
 - o OMC (Open mitral commissurotomy) – Cardiopulmonary bypass needed
 - o MVR – Mechanical, bioprosthetic

Q. Swan-Ganz (Gauge) catheter – (Basic invasive cardiology, F Rahman, P.40)

Full name: Swan-Ganz Balloon-tipped Flow Directed Multichannel Haemodynamic Monitoring Catheter with Thermistor Connector.

Short name: Swan-Ganz invasive Haemodynamic Monitoring Catheter

Parts: Distal end, body, proximal end with 4 channels with port & regulator, ±TPM facility (latest)
Length: 110 cm; Size: 6-7 Fr

Use or indications:

- (Volume status, classify shock, Rx purpose) (Gaffar sir)
- Acute haemodynamic monitoring of cardiac patient (PCW and cardiac index) in CCU / ICU for –
To classify heart failure, etiology of HF, medication & fluid, post-operative care
- General right heart catheterization as flow directed catheter

Parameters can be measured:

- CVP
- PAWP/ PCWP
- LVEDP
- LV filling pressure
- LA pressure
- PAOP (Pulmonary arteriolar occluding pressure)

Conditions can be diagnosed/ evaluated:

- Pericardial tamponade
- Pulmonary hypertension
- RCM

Q. Pulmonary artery wedge pressure (PAWP) or PCWP –

Measure by right heart catheter – Swan-Ganz catheter, balloon tipped with pressure sensor;

- PAWP = LA systolic pressure = LVEDP (LV end-diastolic pressure)
(provided MV is normal)

Pulmonary veins have no valves. (Like IJV)

Q. Central venous pressure (CVP) – indicates RA pressure.

Clinically JVP reflects the right-sided pressure.

Q. Classify shock according to CVP and PAWP –

CVP & PAWP	Conditions
↓↓CVP & ↓↓PAWP	Hypovolaemic shock
↑CVP & ↑PAWP	Volume overload, Acute LVF, CCF, Constrictive Pericarditis
↑↑CVP & ↓↓PAWP (shock with dry lungs)	RHF, RV infarction, Massive PE

Q. Clinically right-sided pressure – by JVP
Clinically left-sided pressure – by Pulse

Q. How to differentiate JVP from carotid pulsation – (Raghawa Rao & Macleod's)

JVP	Carotid pulsation
1. Superficial & lateral in the neck	1. Deeper & medial in the neck
2. Better seen than felt. (Impalpable)	2. Better felt than seen. (Palpable)
3. Has two peaks and two trough per cardiac cycle. (Two peaks per heartbeat in sinus rhythm)	3. Has single upstroke. (One peak per heartbeat)
Rapid inward movement (Macleod)	Rapid outward movement
Pulsation diminished/ abolished by digital pressure at the root of the neck	Pulsation unaffected by pressure at the root of the neck (No effect)
Height of pulsation varies with respiration (falls during inspiration) (x and y descent more prominent during inspiration, a↓ & v↑ transiently during expiration)	Independent of respiration (No effect)
Varies with position/posture of patient (Mean venous pressure falls and JVP becomes less prominent in upright posture)	Independent of position/ posture of patient
Abdominal compression elevates the JV pressure transiently	No effect
Descent more obvious than crests	Upstroke brisker and visible than descent

Q. Measurement of jugular venous pressure – (Raghawa Rao, p.263)

- In adult with normal chest, the sternal angle or angle of Louis is approximately 5 cm above the centre of RA (relatively constant)
- One horizontal is drawn (in relation to the ground by using a scale) at the top level of the venous column and another at the sternal angle
- The vertical distance in centimeters (use another scale) between the two horizontals, that is, from the top of the oscillating venous column in the IJV to the sternal angle will give the JV pressure (which reflects the mean RA pressure)
- Normally, the JV pressure does not exceed 4 cm above the sternal angle, which corresponds to a CVP (RA mean pressure) of 9 cm, since the RA is approximately 5cm below the sternal angle (4+5=9cm).
- **1 mmHg = 13.6 mmH₂O = 1.36 cmH₂O** (পারদ পানির চেয়ে ১৩.৬ গুন ভারী)
- Normal RA mean pressure does not exceed 9 cmH₂O = 9/1.36 = 6.62 ≈ 7 mmHg

Elevated JV pressure: JV pressure > 4 cm above the sterna angle reflects an increase RA pressure.

Q. Jugular venous pulse (JVP) – waves of JVP

- We see two things – 1) Pressure 2) Pulse waves
- Starts with diastolic wave (a), ends with diastolic wave (v)
- We can see only two positive waves (diastolic 'a' wave, systolic 'v' wave)

Waves	Cardiac cycle
'a' wave	Late diastole (Last rapid filling) (Atrial systole)
'c' wave	Early systole
'x' descent (systolic collapse)	Mid systole
'v' wave	Late systole + (initial part of early diastole)
'y' descent (diastolic collapse)	Early diastole (First rapid filling)

- In normal sinus rhythm, the two venous peaks, the **a and v waves**, approximate to atrial and ventricular systole respectively. (Davidson)
- The **x descent** reflects atrial relaxation and apical displacement of the tricuspid valve ring. (**Systolic Collapse**)
- The **y descent** reflects **atrial emptying/ rapid ventricular filling** in **early diastole**. (**Diastolic Collapse**)

Q. Causes of single systolic wave ('v') in JVP – (Gaffar sir)

- Severe TR
- AF

Q. Causes of different wave patterns of JVP – (Raghawa Rao)

Large a waves	Absent a waves
Tricuspid Stenosis , RA myxomas, tricuspid atresia	Atrial fibrillation
PH, PS, acute PE	Sinus tachycardia
RV cardiomyopathy, RV infarction	
Severe AS, HCM	

Tricuspid Stenosis - raised JVP with a **prominent a wave**, and a **slow y descent** due to the loss of normal rapid right ventricular filling. (Davidson)

Regular cannon waves	Irregular cannon waves
Junctional rhythm	Complete heart block (Gaffar sir)
Ventricular tachycardia (VT) 1:1 retrograde conduction	Classic AV dissociation
	VT
	Ventricular pacing
Isorhythmic AV dissociation	Ventricular ectopics
	IVR (Gaffar sir)

Prominent x descent (Systolic collapse) (RV contracts vigorously)	Absent x descent
Constrictive Pericarditis	TR
Cardiac tamponade (Slow y descent)	
ASD	

Prominent v waves	Diminished v waves
Tricuspid regurgitation (TR)	Hypovolemia
Large ASD	Use of nitrates
Gerbode's defect	
Severe congestive heart failure	
Atrial fibrillation	
Cor pulmonale	

Tricuspid regurgitation produces **giant v waves** (a cv wave replaces the normal x descent) that coincide with ventricular systole. (Davidson)

Rapid y descent (Diastolic collapse)	Slow y descent
Severe tricuspid regurgitation	Tricuspid stenosis
Constrictive Pericarditis (rapid ventricular filling in early diastole) (Griffin)	Right atrial myxoma
Severe right ventricular failure	Pericardial tamponade (also prominent x descent)
ASD with mitral regurgitation	

- **Constrictive Pericarditis** – Elevated JVP with a rapid y descent (Davidson)
- **Pericardial tamponade** – Prominent x descent but absent/slow y descent (Gaffar sir)

Q. Causes of acute elevation of JVP – (Gaffar sir)

1. Acute massive pulmonary embolism
2. RV infarction
3. Cardiac tamponade/ pericardial tamponade
4. Tension pneumothorax

Q. JVP waves in Constrictive Pericarditis vs Cardiac tamponade –

Pericardial tamponade – Prominent x descent but absent/ slow y descent

Constrictive Pericarditis – rapid y descent (corresponds with pericardial knock)

Q. Diastolic dysfunction – (ASE/EACVI Diastolic dysfunction Guidelines 2016)

- **Mitral E velocity:** E-wave velocity reflects the LA-LV pressure gradient during **early diastole** & is affected by alteration in the rate of LV relaxation and LAP.
- **Mitral A velocity:** A-wave velocity reflects the LA-LV pressure gradient during **late diastole** & is affected by LV compliance and LA contractile function.
Limitation:
 - Sinus tachycardia, first-degree HB and paced rhythm can result in **fusion of the E & A waves**.
 - Not applicable in AF/atrial flutter patients.
 - Age-dependent. (↑ with aging)
- **Mitral E/A ratio:** Mitral inflow E/A ratio and DT are used to identify the **filling patterns: normal, impaired relaxation, pseudonormal and restricted filling**.
- **Mitral E/e' ratio:** e' velocity can be used to correct for the effect of LV relaxation on mitral E velocity, and E/e' ratio can be used to predict **LV filling pressure**.

- **E/e' ratio <8:** Normal LV filling pressure
- **E/e' ratio >14:** Increased LV filling pressure
- **LAVI:** LA volume reflects the cumulative effects of increased LV filling pressures over time.
- **Pulsed-wave TDI e' velocity (cm/s):** A4CH view, PW Doppler sample volume (usually 5-10 mm axial size) at **lateral & septal basal regions** so average e' velocity can be computed.
Analysis: Peak modal velocity in early diastole at the leading edge of spectral waveform.

In patients with normal LVEF –

The **four** recommended **variables** and their abnormal cutoff values are

1. Annular e' velocity (septal e' <7 cm/sec, lateral e' <10 cm/sec),
 2. Average E/e' ratio >14,
 3. LA maximum volume index (LAVI) >34 mL/m², and
 4. Peak TR velocity >2.8 m/sec.
- (It is recognized that at times only the lateral e' or septal e' velocity is available and clinically valid and in these circumstances a lateral E/e' ratio >13 or a septal E/e' >15 is considered abnormal.)
- >50% positive: DIASTOLIC DYSFUNCTION
 - 50% positive: INDETERMINATE
 - <50% positive: NORMAL diastolic function
- **Grade I diastolic dysfunction:** $E/A \leq 0.8$ + $E \leq 50\text{cm/s}$
 - **Grade II diastolic dysfunction:** ($E/A \leq 0.8$ + $E > 50\text{cm/s}$) or ($E/A > 0.8$ - < 2)
 - 3 criteria to be evaluated (Average E/e' >14, TR velocity >2.8 m/s, LAVI 34ml/m²)
 - 2 of 3 or 3 of 3 – Negative: Normal LAP, Grade I Diastolic dysfunction
 - 2 of 3 or 3 of 3 – Positive: ↑LAP, Grade II Diastolic dysfunction
 - When only 2 criteria are available
 - 2 negative: Normal LAP, Grade I Diastolic dysfunction
 - 1 positive and 1 negative: **Cannot** determine LAP and Diastolic Dysfunction Grade
 - 2 positive: ↑LAP, Grade II Diastolic dysfunction
 - **Grade III diastolic dysfunction:** $E/A \geq 2$

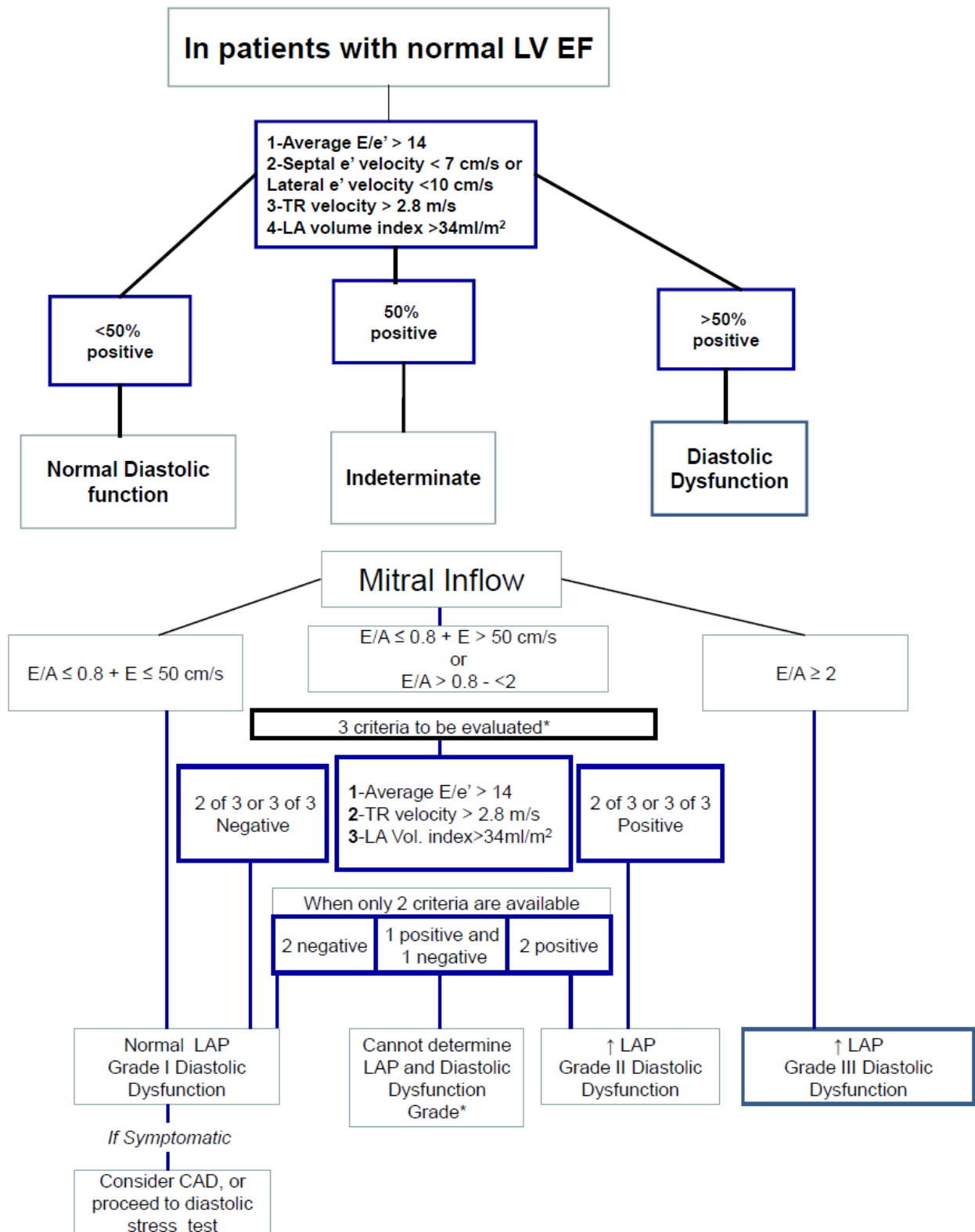


Table 4 LV relaxation, filling pressures and 2D and Doppler findings according to LV diastolic function

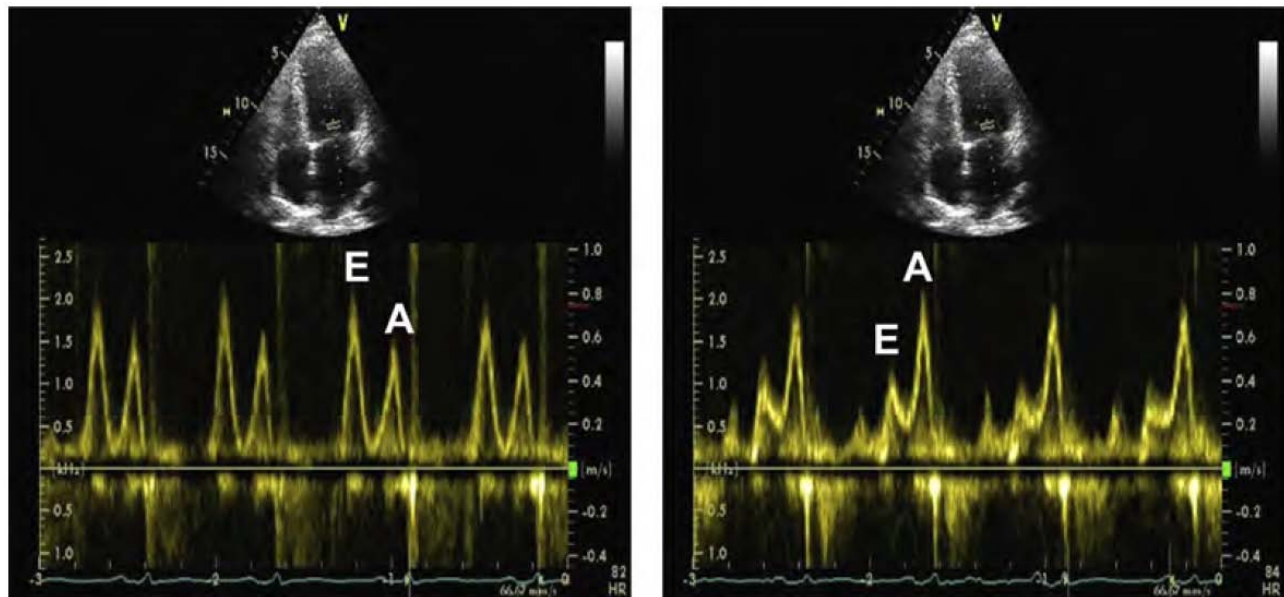
	Normal	Grade I	Grade II	Grade III
LV relaxation	Normal	Impaired	Impaired	Impaired
LAP	Normal	Low or normal	Elevated	Elevated
Mitral E/A ratio	≥ 0.8	≤ 0.8	>0.8 to <2	>2
Average E/e' ratio	<10	<10	10–14	>14
Peak TR velocity (m/sec)	<2.8	<2.8	>2.8	>2.8
LA volume index	Normal	Normal or increased	Increased	Increased

Pseudonormalization

- **Valsalva** (To differentiate from normal) reverse in case of pseudonormalization, proportionally decrease in normal diastolic function.
(Impaired relaxation pattern with Valsalva)

The Valsalva maneuver can help distinguish normal LV filling from pseudonormal filling (and whether restrictive LV filling is reversible or not) because a decrease in E/A ratio of $\geq 50\%$, not caused by E and A velocities fusion, is highly specific for increased LV filling pressures and supports the presence of diastolic dysfunction.

The procedure should be standardized by continuously recording mitral inflow using pulsed-wave Doppler for 10 sec during the straining phase of the maneuver.



Valsalva maneuver in a patient with grade II diastolic dysfunction. At baseline, E/A ratio is 1.3 (left) and decreases to 0.6 (impaired relaxation pattern) with Valsalva.

Q. How to differentiate constrictive pericarditis from restrictive cardiomyopathy – (Amal sir)

	Constrictive pericarditis	Restrictive cardiomyopathy
Pericardium	Thick	Normal
Myocardium	Normal	Thick
Ventricles	Normal (Normal wall thickness)	Obliterated ($\uparrow$ wall thickness)
Atria	Normal (Possible LAE)	Dilated (Biatrial enlargement)
LV function	Normal	Impaired

MV & TV	Normal	Regurgitation
Pericardial knock	Present	Absent
Prominent y descent in JVP	Present	Variable
Equal right- & left-sided filling pressure	Present	Left at least 3-5 mmHg > right
Filling pressure >25 mmHg	Rare	Common
PASP >60 mmHg	No	Common
'Square root' sign (Cardiac cath)	Present	Variable
Respiratory variation in left- and right-sided pressures & flows	Exaggerated	Normal
Septal bounce	Present	Absent
MV inflow	Abrupt halt	Slow relaxation
TD E' velocity	Increased	Reduced

Echo of CP –

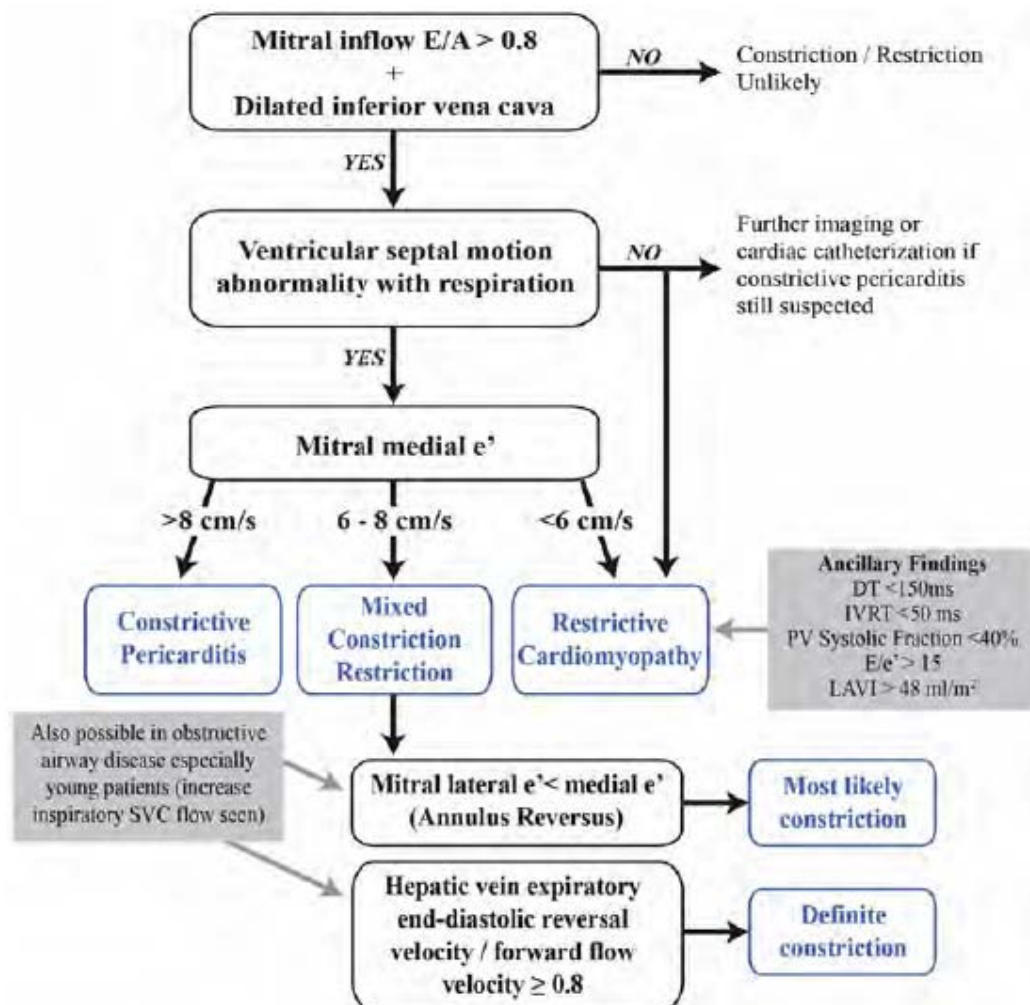
- 2D –
 - Increased echogenicity of the pericardium from thickening, may be effusion (effusive-constrictive)
 - Septal bounce – Abrupt septal shift toward LV in the early diastole and bounce back toward RV following atrial contraction
- M-mode:
 - IVS – Abnormal rapid movements – notching in early diastole
 - LVPW – Abrupt posterior motion in early diastole and flat in remaining diastole
 - IVC & hepatic veins – dilated
 - **Septal bounce 'shudder'** – oscillatory diastolic beat-to-beat movement of the IVS.
 - **Abnormal respirophasic septal shift (Ventricular interdependance)** – Septal movement to the left in early diastole with inspiration whereas in expiration, the IVS shifts back to the right.
(most sensitive echocardiographic findings) (Griffin 5e, p.539)
- Doppler –
 - RV & LV inflow show prominent E wave due to rapid early diastolic filling
 - Short deceleration time of E wave as filling abruptly stops
 - Small A wave as little filling occurs in late diastole following atrial contraction
 - **E/A ratio >2**; DT <160 ms; Isovolemic Relaxation Time (IVRT) <60 ms
 - **TDI**:
 - E/e' ratio should NOT be used to estimate LV filling pressure. (Guideline p.289)
 - **Annulus paradoxus**: E' increases as severity of CP increase (Peak E' ≥ 8cm/s)
Inverse relationship of E/e' compared to PCWP in CP (Griffin)
 - **Annulus reversus**: In CP, the relationship between lateral & medial mitral annuli e' velocities is often reversed. Contrary to normal, e' peak velocity at the **medial annulus (>8cm/s)** is typically higher than peak e' velocity at the lateral annulus.
[Mitral lateral e' < medial e']

Echo of RCM –

- Doppler – Mitral inflow:

Early disease	E<A
Late disease	E>A
IVRT	Constant

- E' decreases as severity increase (Peak $E' < 8 \text{ cm/s}$)
- **Advanced stage of RCM:** (ASE/EACVI Diastolic dysfunction Guideline 2016, p.289)
 - Typical restrictive physiology with a dip and plateau pattern for early diastolic LV pressure changes with time.
 - Mitral inflow E/A ratio > 2.5
 - DT of E velocity $< 150 \text{ msec}$
 - Isovolumic relaxation time (IVRT) $< 50 \text{ msec}$
 - $\downarrow$ Septal & lateral e' velocities ($3\text{--}4 \text{ cm/sec}$) with higher lateral $e' >$ septal e' velocity
 - E/e' ratio > 14
 - Markedly $\uparrow$ LAVI ($> 50 \text{ ml/m}^2$)



Q. Beck's triad – in Cardiac tamponade

- $\uparrow$ JVP
- $\downarrow$ BP
- Muffled heart sounds

Echo – Cardiac tamponade/ pericardial tamponade

- **M-mode: Diastolic collapse of RA & RV.** The RV free wall moves towards the IVS in diastole (normally moves away from IVS), obliterating the RV cavity.

- **2D:** AML shows pseudo-SAM and pseudo-MVP –
 - Pseudo-SAM occurs during anterior swing of the heart
 - Pseudo-MVP occurs during posterior swing of the heart

Q. ECG interpretation – start with p wave in Lead I (Gaffar sir)

p wave should be upright except true/ technical dextrocardia.

- If any limb lead (suppose lead III) shows 'straight line', repeat ECG. (Wadud sir)

Normal 'p wave' –

- Amplitude 2.5mm in limb leads, 1.5mm in precordial leads;
- Duration < 0.1sec (2.5 small squares)

Pathological Q wave –

- > 1 small square wide (>0.03 sec)- (Wadud sir)
- Amplitude $\geq 25\%$ of corresponding R wave
- Found in 2 contiguous leads

If a q wave (false positive) is suspected, ECG should be taken at the height of inspiration.

- Q wave disappear over time in **25% of inferior MI**, **12% of anterior MI** (Wadud sir)

Q. Physiological septal q wave found in – lateral leads (I, aVL, V5-6)

Q. Normal sinus rhythm –

- Every QRS complex is preceded by p wave
- P wave is upright in Lead I & II

Q. Apparently normal ECG, following possibilities should be ruled out – (Wadud sir)

1. 1st degree heart block
2. WPW syndrome
3. True posterior MI
4. Brugada syndrome
5. Long QT syndrome
6. Pacing ECG (Atrial)

Q. LBBB pattern ECG, but positive complex in V₅₋₆ – (Wadud sir)

- Look for small spikes in anterior leads
- Possibility of RV pacing

Q. 'Q wave' may disappear following acute STEMI – (Wadud sir)

- 25% cases after inferior MI
- 12% cases after anterior MI

Q. Causes of 'poor progression of R wave' – (R wave height $\leq 3\text{mm}$ in V₃) LITFL.com

- **Prior antero-septal or anterior MI**
- **LVH**
- **DCM**
- LBBB, LAHB
- Inaccurate lead placement (e.g. transposition of V1 & V3)

- COPD, left-sided pneumothorax/ pleural effusion
- Marked clock-wise rotation (When the heart rotates clockwise, the **transition zone**, which is usually V₃ or V₄, **moves toward V₅ or V₆.**) (Late transition)
- Deformity of the chest wall; **thick chest wall (Gaffar sir)**
- **Female (Gaffar sir)**
- Normal variant

Dextrocardia (Reverse progression of R wave)

Q. During precordium exam – say S1 is normal (don't say - 'S1 is normal in all areas')

Q. Haemodynamic effects of respiration – (Davidson)

	Inspiration	Expiration
JVP	Falls	Rises
BP	Falls (upto 10 mmHg)	Rises
HR	Accelerates	Slows
S2	Splits	Fuses
VR	Increases	Decreases
SV	Decreases	

Inspiration prolongs RV ejection, delaying P2, and shortens LV ejection, bringing forward A2; Expiration produces the opposite effects.

- **During inspiration**, ↑VR → ↑RV volume → Blood goes to lungs (capacitance) → ↓LV filling → ↓EF (But ↑HR to maintain CO)
- **During expiration**, ↓VR but ↑LV filling → ↑SV (But ↓HR to maintain CO)
- **Septal interdependence** – IVS works as part of dominant ventricle

Q. Pulsus paradoxus – (Davidson)

- Exaggerated fall in BP during inspiration
- Cardiac tamponade & severe airways obstruction.
- In airways obstruction, it is due to accentuation of the change in intrathoracic pressure with respiration.
- In cardiac tamponade, compression of the right heart prevents the normal increase in flow through the right heart on inspiration, which exaggerates the usual drop in venous return to the left heart and produces a marked fall in BP (> 10 mmHg fall during inspiration). (Davidson)
- During inspiration, ↑VR → RV enlarges → IVS pushes into LV → **↓LV filling** & Lung expands → Pulmonary vasculature expands → More blood pools in lungs → **↓LV filling**, finally **↓SV and ↓pulse amplitude** (Net + Gaffar sir)
- Initially pulse feeble, but at the height of inspiration, pulse will be absent.

Q. Bedside examination in pt with long-standing hypertension: (Gaffar sir)

Precordium – Forceful, sustained apex,

Loud A2, reverse or narrow splitting

Echo – Concentric LVH

Q. ECG criteria of LVH: 14, 11, 20 (Gaffar sir) LITFL.com (Voltage criteria of LVH)

- R in aVR ≥ 14 mm
- R in aVL ≥ 11 mm (**Sokolow-Lyon criteria**)

- R in aVF ≥ 20 mm
- R in lead I + S in lead III > 25 mm
- R in V₄, V₅ or V₆ > 26 mm
- SV₁ + RV₅/V₆ > 35 mm
- Largest R + largest S in precordial leads > 45 mm

Most commonly used **Sokolow-Lyon criteria**: (Baltazar, p.71)

- SV₁ + RV₅/V₆ > 35 mm
- R in aVL > 11 mm

Other criteria: (Baltazar, p.71)

- **Romhilt and Estes**: Score ≥ 5 points = LVH, Score 4 points = Probable LVH
- **Cornell voltage criteria**: R in aVL + S in V₃ > 28 mm in men & > 20 mm in women (Gaffar sir)
- Cornell product
- Total QRS voltage
- In presence of LBBB: SV₁ + RV₅/V₆ > 45 mm

Q. ECG criteria of RVH: (Khalequzzaman sir)

- R $>$ S in V₁
- RAD
- R > 7 mm
- In presence of complete RBBB: RAD + R > 15 mm in V₁
- In presence of incomplete RBBB: R > 10 mm in V₁

Q. ECG criteria of LAHB (Left anterior hemiblock): (Khalequzzaman sir)

- LAD
- rS (**S**mall r, deep S) in lead II, III, aVF (in contrast, in Inf. MI, only Q wave)

Q. Causes of pressure overload –

Left side:	Right side:
• AS	• PS
• HOCM	• Pulmonary Hypertension
• Systemic hypertension	
• CoA	

Pressure overload causes **hypertrophy**, unless systolic dysfunction causes dilatation in late stage.

Q. Causes of LV volume overload – (LVH without strain)

- MR, AR
- In young patients – VSD, PDA (Not ASD)

Volume overload causes **dilatation**, not hypertrophy.

Q. Causes of RV volume overload – (Luthra p.130)

- ASD (from LA)
- TR (from RA)

- PR (from PA)
- VSD (from LV)
- Rupture of Sinus of Valsalva (SOV) (from Aortic root)

Q. ECG feature of LVH due to volume overload & pressure overload – (Baltazar, p.70)

- **Pressure overload** – LVH (Voltage criteria), T inversion
- **Volume overload** –
 - Prominent Q waves,
 - Tall R waves,
 - Tall and upright T waves in V₅₋₆,

Q represents LV end diastolic point. At this point, LV is maximally stretched, filled and stressed. Maximum LVID corresponds to q wave.

Q. Normal cardiac apex – (Raghawa Rao, 2e p.297) (Nazrul sir)

- Outermost & lowermost point of maximum impulse (PMI) in **early systole**,
- which imparts a perpendicular thrust to a palpating finger,
- followed by a slight medial retraction in the **late systole** (better seen than felt).
- Located in the 4th or 5th left intercostal space at or inside the mid-clavicular line,
- ≤10 cm from the mid-sternal line.
- Confined to one intercostal space, which is <3cm in diameter, and
- Lasts for <50% of systole.

Q. Splitting of S2 – occur only during inspiration, normal or narrow splitting
Only during expiration – reverse splitting

Both inspiration & expiration – **wide & fixed** (as in ASD)

Fixed – Because same amount of blood exit RVOT during both phases of respiration.
~~because equalization of pressure of LA & RA in both phases of respiration.~~

Wide – because **delay in RV ejection** (↑SV & RBBB). (Davidson)
Increased flow through TV (Nazrul sir).

ASD with PH – Relatively wide & fixed splitting of S₂ (Wadud sir)

Q. Causes of reverse splitting of S2 (only during expiration)

1. **(Complete) LBBB** (Raghwa Rao)
2. **LVOT obstruction – HOCM, Severe AS**
3. **Severe systemic hypertension**
4. **RV pacing**, RV ectopics
5. PDA, Post-stenotic dilatation of aorta secondary to AS, AR

Q. ECG findings of acute pericarditis – stages (Griffin 5e, p.523)

- ECG changes (upto 60% of cases) evolve through four stages.
- These may be absent in TB and neoplastic pericarditis.
- In large effusion, ECG may show **electrical alternans or low voltage or both**.
- **Stage I –**
 - Diffuse ST segment elevation (usually upsloping) with concavity upwards,
 - **Upright T waves** and
 - **PR segment depression** in all leads except **V₁ and aVR**.

- o Often reciprocal PR segment elevation with ST depression in aVR (**Knuckle sign**) (**'PR-ST discordant sign'**) (Gaffar sir)
- Stage II (several days later) –
 - o Resolution of previous PR- and ST-changes ST elevation resolves,
 - o **Flattening of T waves**
- Stage III – ST isoelectric, **T wave inversion**
- Stage IV (after several days to weeks) – ST isoelectric, **upright T waves again**.

ECG of Acute STEMI: (Gaffar sir)

- ST elevation with convexity upwards.
- Following coronary territory (Diffuse ST elevation in acute pericarditis)
- T wave inversion (Upright T wave in Stage I of acute pericarditis)

Q. Examination of pericardial rub – (Gaffar sir)

- Found in <33% cases (Griffin), (Found in 1/3 cases) (Wadud sir)
- Scratchy and high-pitched sound, often evanescent with changes in quality and intensity on serial exam.
- **Sitting leaning forward, breathe hold in expiration** (Gaffar sir)
(Best heard during inspiration) – (Griffin 5e, p.523)
- Auscultate in left parasternal area (3rd-4th space)/ **Bare area of heart** (other names of this area: tricuspid area/ Neo-aortic area) with the diaphragm of stethoscope
- Dynamic auscultation: **Knee-elbow position** → pericardial rub becomes prominent
- Chest pain of acute pericarditis – improved by sitting up and leaning forwards. (Griffin)
- Pericardial rub – better heard in uraemic Pericarditis, due to ↑fibrin deposition (Wadud sir)

Q. If pericardial rub disappears, possibilities – (Gaffar sir)

- Recovery of acute Pericarditis
- Development of pericardial effusion

Q. Diastolic sound (after S₂) – causes (Gaffar sir)

- S₃ (Volume overload)
- S₄ (Pressure overload)
- Pericardial knock (Constrictive Pericarditis) (earlier than S₃, heard with diaphragm, higher frequency)
- Opening snap (Mitral stenosis)
- Tumour plop (Prolapsing LA myxoma)
- **Early diastolic sound:** OS, tumour plops, pericardial knock, opening sounds of prosthetic valve
- Mid diastolic sound: S₃
- Late diastolic sound: S₄

Q. Acute STEMI Inferior with AF, site of lesion –

- Proximal RCA

Q. Acute STEMI inferior with first-degree AV-block, Mobitz type I –

- RCA, AV nodal branch

Q. Acute STEMI inferior with first-degree AV-block, no RVI –

- RCA, distal to RV branch, proximal to AV nodal branch

Q. Acute STEMI inferior with posterior extension – means

- RCA dominant

Q. Site of lesion in AV block– (Gaffar sir)

Heart block	Site of lesion
First degree HB	AV node
Mobitz type I Second degree HB	AV node (Gaffar sir) (Baltazar), Infranodal – Intra-Hisial / Infra-Hisial (Baltazar)
Mobitz type II Second degree HB	Always infranodal (His-Purkinje system) Bundle of His or below (Intra-/ infra-Hisial)
CHB following inferior MI	AV node
CHB following anterior MI	Below AV node (Infranodal)

	AV nodal block	Infranodal block
Cause	Acute MI inferior	Acute MI anterior
Course	Usually reversible, PPM often not needed	Progressive, permanent. PPM often needed
Comes to the rescue	AV junction, Rate 40-60/min	Ventricular escape rhythm, Inherently slower rate 20-40/min
Response to atropine	Yes	No
QRS complex	Narrow	Wide
Force of contraction	More effective than ventricular impulse	Ventricles do not contract simultaneously, ineffective beat, low CO than junctional escape complex

Q. In inferior MI, if vagolytic (atropine) given - ↑HR

Usually in inferior MI → intense vagal stimulation.

- 1 ample Atropine, every 3-5 min until a desired HR is achieved (Baltazar – 0.5mg)
- Complete vagal blockade is expected when a total dose of 3 mg iv over 2h
- Atropine should not be given in doses smaller than 0.5mg because small doses stimulate the vagal nuclei and may enhance parasympathetic activity, resulting in paradoxical slowing and worsening of the AV block
- Atropine is not effective in infranodal AV block

Q. Virtual description of CAG findings of Acute STEMI Inferior with posterior extension –

RCA – dominant vessel, 80-90% stenosis in proximal RCA, proximal to RV branch

Q. Polymorphic VT –

- Regular (form of) PVT – Ischaemic (Acute STEMI inferior)
- TdP – when the PVT is associated with prolonged QT interval
- **All TdP are PVT, but all PVT are not TdP.**

Q. TdP – PVT preceded by long QT

- VT with twisting of axis (Wadud sir)

Q. Bidirectional VT – not preceded by long QT

- Frequently associated with **digitalis toxicity**
- VT originates from two separate locations, the left anterior and left posterior fascicles of the LV. Thus the VT has **RBBB configuration** and the axis of the ectopic impulse alternates between right & left axis in the frontal plane (Baltazar)
- When the ectopic impulse originates from the left anterior fascicle, the QRS complex is deviated to right. (Baltazar)
- When the ectopic impulse originates from the left posterior fascicle, the QRS complex is deviated to left. (Baltazar)
- **Catecholaminergic PVT (CPVT)** – may be polymorphic, **bidirectional**, less commonly VF.

Q. Corrected QT interval – (Wadud sir)

Upto 440 ms (Male), Upto 460 ms (Female)

History taking from a patient with long QT interval –

- H/O syncope, unconsciousness
- Family H/O premature SCD
- H/O vomiting, diarrhoea, diuretics

Q. Drug causes of long QT interval – (Wadud sir)

Anti-arrhythmics	Anesthetics/ antiasthmatics	Antibiotics	Antihistamines	CNS active drugs
Quinidine (Ia)	Droperidol	Clarithromycin	Terfenadine	Droperidol
Procainamide HCl (Ia)	Adrenaline	Erythromycin	Diphenhydramine	Haloperidol
Disopyramide PO ₄ (Ia)		Pentamidine		Pimozide
Propafenone (Ic)		Trimethoprim- sulfamethoxazole		Risperidone
Flecainide (Ic)		Ketoconazole		
Amiodarone (III)		Fluconazole		
Sotalol HCl (III)	Gastrointestinal stimulants	Antihyperlipidemic		
Ibutilide Fumarate	Cisapride	Probucol		
Dofetilide (III)				

Hurst the heart, 14e, p.1345

- Ranolazine can prolong QT interval. (Wadud sir)
- Long QT syndrome subtypes have different triggers, which are important when counselling patients.
- Adrenergic stimulation (e.g. exercise, emotion, swimming- Hurst) is a common trigger in long QT type 1.
- A sudden noise (e.g. an alarm clock) may trigger arrhythmias in long QT type 2. (Auditory)
- Arrhythmias are more common during **sleep** in type 3.

18.34 Causes of long QT interval and torsades de pointes
Bradycardia
Bradycardia compounds other factors that cause torsades de pointes
Electrolyte disturbance
- Hypokalaemia - Hypomagnesaemia - Hypocalcaemia

Drugs
- Disopyramide, flecainide (and other class Ia, Ic anti-arrhythmic drugs, p. 574) - Sotalol, amiodarone (and other class III anti-arrhythmic drugs) - Amitriptyline (and other tricyclic antidepressants) - Chlorpromazine (and other phenothiazines) - Erythromycin (and other macrolides) ... and many more
Congenital syndromes
- Long QT1: gene affected <i>KCNQ1</i>: K⁺ channel, 30–35% - Long QT2: gene affected <i>HERG</i>: K⁺ channel, 25–30% - Long QT3: gene affected <i>SCN5A</i>: Na⁺ channel, 5–10% - Long QT4–12: rare

TABLE 55–16. Genotype–Phenotype Correlation in Long-QT Syndromes

Phenotype	Gene	T Wave	Trigger
LQT1	<i>KCNQ1</i>	Early onset, broad-base T	Emotion, swimming
LQT2	<i>HERG</i>	Low amplitude	Auditory
LQT3	<i>SCN5A</i>	Late T, normal amplitude	Sleep

Q. Long QT syndrome –

- QT normally <440 ms in male, <460 ms in female
- QT best seen in lead II, V₅₋₆
- QTc (Bazett's formula) = $QT / \sqrt{RR}$
- Congenital Long QT syndrome – due to sympathetic overactivity;
Rx – β -blocker, ICD.
LQT 1-12 types (H/O deafness, FH of SCD)
- Acquired Long QT syndrome – bradycardia induced; Rx – Isoprenaline
Rx modalities – MgSO₄ IV bd,

Q. Drug Rx of TdP/ Torsades de pointes (Polymorphic VT + long QT) –

- IV magnesium** (8 mmol over 15 mins, then 72 mmol over 24h)
(Beximco - IV Nalepsin 4%, 100 ml, Renata- Magsum 100ml, 2.5 g/5ml)
- IV Isoprenaline** (reasonable alternative to pacing, but **should be avoided in congenital LQTS**)
- Overdrive suppression/ Atrial overdrive pacing** (TPM – fix rate at 100/m to prevent bradycardia induced TdP) (Gaffar sir)
- DC shock
- β -blockers** – to prevent syncope in congenital LQTS (Cardiac Na or K channel mutations)
- ICD in extreme QT interval prolongation (>500ms) or certain high risk genotype
- Left stellate ganglion block in resistant arrhythmias

Q. Inj. Calcium gluconate (Rule of 10)

10%, 10 ml, over 10 min

Q. Pt in shock, we can use Dopamine along with Ivabradine to control rate.

Q. Forrester classification – PAWP and Cardiac index – (Griffin)

(Clinically BP and Lung base, JVP)

Subsets

I – PCWP <18, CI >2.2 mortality- 03%

II – PCWP >18, CI >2.2 mortality- 09%

III – PCWP <18, CI <2.2 mortality- 23%

IV – PCWP >18, CI <2.2 mortality- 58%

Q. Define Cardiogenic shock –

- Inadequate tissue perfusion
- Reduction of cardiac output (hypotension) despite adequate filling pressure
- Autopsy studies have revealed that **at least 40% of the LV** is affected in most patients presenting with cardiogenic shock complicating acute MI. (Wadud sir)

(SBP <100 mmHg in inferior MI with RVI → Fluid challenge,
SBP <90 mmHg → Cardiogenic shock) (Wadud sir)

Q. Inotropes –

- **Cardiogenic shock –**
 - **Noradrenaline** (NA),
 - **Dopamine** if inadequate response to NA along with reducing dose of NA
- Cardiogenic shock with low CO (Harrison)
 - SBP >100 : GTN 10-20 µg/min IV
 - SBP 70-100, No S/S of shock: Dobutamine 2-20 µg/Kg/min IV
 - SBP 70-100, S/S of shock: Dopamine 2-20 µg/Kg/min IV
 - SBP <70: NA 0.5-30 µg/min IV
- **RVI – IV fluid, Dobutamine**
- Septic shock – **Noradrenaline** (NOT dobutamine as it causes splanchnic vasodilation) + IV fluid + systemic antibiotics
- Anaphylaxis – S/C Adrenaline
- NA – No effect on HR, doesn't ↑ cardiac workload
- Adrenaline – ↑ Cardiac workload & O₂ demand, ↑ HR
- Dopamine – mixed effect, inotrope + vasopressor

Oral inotrope – Pseudoephedrine

DOBUTAMINE: (Gaffar sir)

- Indication: RV infarction (↓ RV afterload by pulmonary vasodilation)
- Contraindication: Septic shock (Dobutamine causes splanchnic vasodilation), tachyarrhythmias
- Acute STEMI inferior with RVI with CHB – if TPM cannot be done instantly, dobutamine can be given as bridging therapy to ↑ HR as well as to ↑ BP.
(In CHB, Isoprenaline, if not in MI settings)

Dobutamine: (Drugs for the heart, p.188)

- A synthetic analog of dopamine
- Competitive β-adrenergic stimulating agent (**β₁ > β₂ >>>> α**)
- Potent inotropic effect
- Can be infused for up to 72 hours with monitoring
- Used in stress echocardiography
- Check blood potassium to minimize arrhythmias

- **Dobutamine dose:** 2.5-10 µg/kg/min, with lower doses (2.5-5 µg/kg/min) frequently sufficient.
- **Inj. Dobutamine 250mg/5ml, 1 amp + 45 ml 5%D/A, in syringe pump @3.6ml/hr**
(50 ml=250mgx1000= 250000µg, 1 ml=5000µg,
For a 60 kg patient, **5µg/kg/min**, 5x60x60=18000µg/hr=3.6ml/hr)
- **Inj. Dobutamine 250mg/5ml, 2 amp +90 ml NS, @8-15µd/min (10-20µg/kg/min)**
 - o 2 amp=10ml=500mg, 2 amp + 90 ml NS= 100 ml= 500mg= 500000 µg,
 - o 1 ml= 15d= 60µd= 5000µg,
 - o In a 60 kg patient, 20x60 µg/min= 1200µg/min,
 - o 60µd= 5000µg, 30µd= 2500µg, 15µd= 1250µg,
 - o 15µd/min= 20µg/min, 8µd/min= 10µg/min (Chinmoy)

Q. Relative selectivity of adrenoceptor agonists – Relative receptor affinities

Ref (Katzung)	Relative receptor affinities
Alpha agonist:	
Clonidine	$\alpha_2 > \alpha_1 >>>> \beta$
Mixed α- & β-agonists:	
Norepinephrine/ Noradrenaline	$\alpha_2 = \alpha_1, \beta_1 >> \beta_2$
Epinephrine/ Adrenaline	$\alpha_2 = \alpha_1, \beta_1 = \beta_2$
β-agonists:	
Dobutamine	$\beta_1 > \beta_2 >>>> \alpha$
Dopamine agonists:	
Dopamine	$D1=D2 >> \beta >> \alpha$

Q. Causes of (Cardiogenic) shock after acute extensive anterior MI – (Gaffar sir)

- Severe LV systolic dysfunction
- Ventricular septum rupture (VSR)
- Acute MR
- Drugs – Nitrate, β -blocker, ACEi
- Arrhythmias

Q. Anterior MI with bradycardia – (Gaffar sir)

- CHB
- SA node supplied by Left system

Q. How will you follow up a patient following Acute STEMI anterior – (Gaffar sir)

- Symptoms – chest pain, SOB, urine output, lightheadedness (Low BP, postural hypotension)
- Signs –
 - o Pulse rate:
 - Tachycardia
(Ongoing ischaemia, covert/ overt HF, fever, other causes)
 - Tachy-arrhythmias
 - o Pulse volume: Lesser volume, more myocardial damage.
 - High volume in hypertensive emergency.
 - o Systolic BP: Usually low. (correlates with degree of myocardial damage)
 - Cardiogenic shock
 - o JVP: Normal (Acute $\uparrow$ JVP in RVI)

Precordium:

- Inspection: Unremarkable
- Palpation:
 - o Apex beat: Located normally/ shifted down & out (correlate with degree of myocardial damage)
 - o Character: Normal or **SOFT**
(Softness correlates with degree of myocardial damage)
 - o **Left parasternal lift**: due to LA enlargement & LV volume overload
(**Not left parasternal heave** which is due to RV pressure overload)
 - o Thrill: Systolic thrill in mitral area or left lower parasternal area
- Auscultation:
 - o S1 – soft
 - o S2 – usually normal
 - o S3 gallop (Tricuspid area), S4 gallop, Summation gallop
 - o Murmurs:
 - PSM in apex (Acute MR),
 - PSM in left parasternal area (VSR),
 - o Pericardial rub in bare area of heart
 - o Lung bases (Acute LVF)

Q. Junctional rhythm/ nodal rhythm – (Gaffar sir)

- Retrograde p wave may occur before or after QRS (inverted in inferior leads), or absent p wave (buried in the QRS complex), Normal QRS complex
- Junctional bradycardia: < 40 /min
- Junctional rhythm: 40-60/min
- Accelerated junctional rhythm: 60-100/min
- Junctional tachycardia: > 100/min

Q. Atrial ectopic Rx

- Propantheline + Theophylline
(to stimulate SA node/ native pacemaker).
- NOT β -blocker

Q. AF vs Atrial ectopic –

AF: Pulse deficit, S1 variable, persist on exercise, JVP – absent a wave

Atrial ectopic: No pulse deficit, S1 not variable, disappear on exercise

Q. Causes of AV dissociation –

1. Junctional escape rhythm (Rate 40-60/m)
2. Accelerated junctional rhythm (Rate >60-100/m)
3. Junctional tachycardia (Rate >100/m)
4. Ventricular escape rhythm (Rate 15-40/m)
5. **AIVR** (Rate >40-100/m)
6. **VT** (Rate >100/m)
7. **Ventricular paced rhythm** (Single chamber ventricular pacing, TPM)
8. **CHB**

Q. Clinical features of AV dissociation –

- Variable S1
- Irregular Cannon waves in JVP
- ECG – variable PR interval

Q. Criteria/ triad of LBBB – (Baltazar)

- Wide QRS (≥ 0.12 s)
- Broad monophasic R wave in Lead I, V_{5-6} ; Dominant S wave in V_1 (RSR' pattern in V_{5-6} , I, aVL)
- Absence of Q waves in lead I, V_{5-6} (Small Q waves are still allowed in aVL)

Associated features:

- **Appropriate discordance** (displacement of ST segment & T wave in an opposite direction to the dominant deflection of the QRS complex)
- Poor R wave progression in the chest leads
- RS complex rather than monophasic complex in leads V_{5-6}
- LAD – common but not invariable finding

Explanation: LITFL.com

- **Normally** IVS is activated from **left to right**, producing **Q waves in the lateral leads**.
- **In LBBB**, the normal direction of IVS depolarization is **reversed** (becomes **right to left**) as the impulse spreads first to the RV via the RBB and then to the LV via the IVS.
- This sequence of activation extends the QRS duration to ≥ 120 ms and eliminates the normal septal Q waves in the lateral leads.
- As the ventricles are activated sequentially (right, then left) rather than simultaneously, this produces a broad or notched ('M'-shaped) R wave in the lateral leads

Q. Approach to a patient with LBBB –

- Typical ischaemic chest pain
- New onset or not
- Cardiac biomarkers / enzymes elevated/ not
- Sgarbossa criteria
- Pseudo-normalization of T wave
- Echo – Jerky, hypokinetic IVS
- CAG

Q. Ischaemic LBBB –

- Pseudo-normalization of T-wave indicate ischaemia
- Presence of even physiological q in LBBB or RBBB indicate ischaemia

Q. LBBB – Echo

Jerky IVS (Jerky hypokinetic IVS if ischaemic LBBB)

Decreased systolic thickening (at least 50%)

Q. Sgarbossa criteria – (Baltazar-358)

1. Concordant ST elevation 1 mm or more (V_{5-6} & often in II, III, aVF) (5)
2. Concordant ST depression 1 mm or more (V_{1-3}) (3)
3. Discordant ST elevation 5 mm or more (in any leads, e.g. V_{1-3}) (2)

Score 3 or more – highly sensitive & specific

Q. Modified Sgarbossa criteria – (Khalequzzaman sir)

3. Discordant ST elevation >25% of the depth of preceding S wave (in any leads, e.g. V₁₋₃)

Cabrera sign:

Champman sign:

Q. Contraindications of GTN/ Nitrates –

1. Acute STEMI Inferior with RVI
2. Hypotension
3. Severe headache
4. Extreme tachy/ bradycardia
5. Hyperdynamic states (anaemia, thyrotoxicosis)
6. Outflow tract obstruction

Q. Digoxin dose/ Dose of IV digoxin – (Inj. Digoxin 0.5 mg = 500 µg)

(1 amp. + 50 ml NS) IV @ 50 µd/min over 1 hour

- o Dose: 0.02 mg/Kg, If weight 50 Kg, then 50x0.02=1mg (Nazrul sir)
 - 50% iv stat (1 amp),
 - after 8 hours 25% (1/2 amp),
 - after 8 hours 25% (1/2 amp).
- o ? Orally – 2+2+2 for 1 day, then 1+1+1 daily or
2+0+2 for 2 day, then 1 tab daily
- o Digoxin toxicity if hypokalaemia
- o Digoxin not very effective in hyponatraemia? (Wadud sir)
- o Digoxin can be given **orally, IV, deep IM** with adequate massage (Nazrul sir)

Q. Contraindications of Digoxin:

1. Outflow tract obstruction (AS, PS, HOCM, TOF)
2. Severe anaemia
3. Thyrotoxicosis (itself hyperdynamic)
4. ACS (can cause free wall rupture, VSR) (Gaffar sir)

Q. Digoxin side effect & digoxin toxicity – (Nazrul sir)

S/E – bradycardia, 1st degree HB

Toxicity – Arrhythmias: frequent PVC, CHB; long QT

Q. Digoxin effect & digoxin toxicity –

Digoxin effect – (Schamroth)

- reverse tick/ sagging ST segment in lateral leads (V₄₋₆, I, aVL) (ST depressed from baseline, concave upwards)

(ST morphology described as slurred/ sagging/ scooped/ reverse tick/correction or check mark/ hockey stick/ Salvador Dali's moustache)

- **Downward slope of the ST segment begins from the isoelectric base** (digoxin in therapeutic dose does not depress the proximal part of the ST segment)
(If the beginning of the ST segment with inverse check/tick mark configuration is already depressed below the isoelectric level, it may be

- Digoxin toxicity
 - Primary cause of ST depression (myocardial ischaemia)
- **Biphasic T wave:** Initial negative deflection with terminal positive deflection (**Terminal positivity of T wave**)
(First part of T wave is typically continuous with the depressed ST segment)

Digoxin toxicity –

- Terminal part of the T wave does not rise above the baseline
(Ischaemia – ST depression)
- Proximal ST depression (Downward slope of the ST segment does not begin from the isoelectric base)

Digoxin-induced cardiac arrhythmias – any abnormal cardiac rhythm except Mobitz type II second-degree Heart block

- First-degree AV block
- Second-degree Heart block of Wenckebach type
- Occasionally CHB, SA block
- Paroxysmal Atrial Tachycardia (PAT) with block (Characteristic)
- Accelerated junctional rhythms with varying degrees of block
- PVC (bigeminy, trigeminy), Unifocal/ Multifocal
(Frequent PVC during AF may suggest digoxin toxicity)
- Bi-directional VT (Other causes – HypoK PP, myocarditis, CAD, idiopathic DCM, Catecholaminergic PVT, herbal aconite poisoning, metastatic cardiac tumors)

Q. Target HR in MS:

- Resting HR: 70-90/min (average 80/min)
- Exertional HR: 90-110/min (average 80/min)
- Digoxin acts on resting HR
- β -blocker acts on both resting & exertional HR.

Q. Causes of hypotension with bradycardia – (Gaffar sir) (Nazrul sir)

- Acute STEMI Inferior with RVI
- Vasovagal syncope [Reflex (neurally mediated) syncope]
 - Orthostatic VVS: standing, less common sitting
 - Emotional: fear, pain (somatic or visceral), instrumentation, blood phobia
- Drug overdose – β -blocker, rate-limiting CCB

Q. Fluid should be given in which shock – (Gaffar sir)

- Hypovolemic shock
- Inferior MI with RVI
- Septic shock

Q. Classify syncope – (2018 ESC Guidelines)

- Reflex (neurally mediated) syncope –
 - Vasovagal –
 - Situational –

- Carotid sinus syndrome –
- Syncope due to OH –
 - drug-induced – vasodilators, diuretics, phenothiazine, antidepressants
 - volume depletion,
 - primary autonomic failure,
 - secondary autonomic failure.
- Cardiac syncope –
 - Brady- & tachy-arrhythmias –
 - Structural cardiac – AS, AMI/ischaemia, HOCM, cardiac masses (atrial myxoma, tumours etc.), pericardial tamponade, congenital anomalies of coronary artery, prosthetic valve dysfunction
 - Cardiopulmonary & great vessel – PE, acute aortic dissection, PH

Q. Concertina effect / Accordion effect in cath lab – Nazrul sir

Lesions or stenoses that appear after placement of interventional guidewire in a tortuous artery and disappear when the wire is withdrawn are known as Pseudolesions. This effect is known as the accordion effect or Concertina effect.

Q. How to start entresto/ ARNI? (Valsartan–Sacubitril)

Tab. Entresto (Novartis)/ Sabitar (Incepta), 24/26, 49/51, 97/103mg

- Low-dose ACEi, STOP, **wait 36 hours** then switch to ENTRESTO,
- Low-dose ARB or NO ACEi/ARB → Switch to ENTRESTO
 - starting dose 50mg bd as tolerated by the patient, follow up in 2-4 weeks,
 - titrated to 100mg bd as tolerated by the patient, follow up in 2-4 weeks,
 - titrate to target dose 200mg bd as tolerated by the patient.

(Low-dose ACEi: total daily dose of ≤10 mg of enalapril or therapeutically equivalent dose of another ACEi (eg, lisinopril ≤10 mg; ramipril ≤5 mg)

Low-dose ARB: total daily dose of ≤160 mg of valsartan or therapeutically equivalent dose of another ARB (eg, losartan ≤50 mg; olmesartan ≤10 mg)

- High-dose ACEi, STOP, **wait 36 hours** then switch to ENTRESTO,
- Low-dose ARB or NO ACEi/ARB → Switch to ENTRESTO
 - starting dose 100mg bd as tolerated by the patient, follow up in 2-4 weeks,
 - titrate to target dose 200mg bd as tolerated by the patient.

(High-dose ACEi: total daily dose of >10 mg of enalapril or therapeutically equivalent dose of another ACEi (eg, lisinopril >10 mg; ramipril >5 mg)

High-dose ARB: total daily dose of >160 mg of valsartan or therapeutically equivalent dose of another ARB (eg, losartan >50 mg; olmesartan >10 mg)

- **ENTRESTO may be administered with or without food.**
- **With ARNI (Valsartan–Sacubitril), diuretics dose –** Should be reduced.
- This 36 hours interval is to prevent angioedema (Wadud sir)

Q. High lateral MI – due to D1 occlusion

Q. LAD – site of lesion & ECG findings –

- **RBBB – proximal to S1 (septal perforator)**
- Distal to S1 but proximal to D1 - ???
- I, aVL – (D1)
- V1-V4 (Anteroseptal) – distal to D1

Q. True posterior MI – (Gaffar sir)

- Commonly due to LCX occlusion
- RCA occlusion before **large PLV branch** (Inferior MI with posterior extension)

Q. Acute STEMI inferior (Inferior MI) due to occlusion of – (Gaffar sir)

- Right-dominant RCA (PDA from RCA)
- Left-dominant LCX (PDA from LCX)
- Type IV LAD (Antero-inferior MI)

Q. Acute STEMI anterior –

First TPM, then Thrombolysis

Not thrombolysis first (Bleeding can't be controlled from puncture site)

Q. Symptoms of bi/tri-fascicular block –

Syncope, dizziness, vertigo

Rx – TPM>PPM

Q. VSD, but oxygen saturation step-up in RA (rather than RV)

Defect in atrioventricular septum (septal leaflet of MV – above;
to setal leaflet of TV – below)

Gerbode defect in AVS (Shunt LV to RA)

Q. Types of ASD (Atrial septal defects) with association– (F:M= 3:1) (Satpathy)

1. **ASD secundum** (74%) – defect in fossa ovalis; association – **MVP**
 - Small or Restrictive ASD: Pressure difference between two atria
 - Large or Nonrestrictive ASD:
2. **ASD primum** (20%) – defect inferior to fossa ovalis; association – cleft MV
3. **Sinus venosus type** (5%) – defect in upper part of fossa ovalis;
association – TAPVC, PAPVC
4. **Coronary sinus type** (1%) – defect in posterior/ inferior part of fossa ovalis;
association – PLSVC
5. **Common A-V canal**: Complete form of endocardial cushion defect (ASD + VSD)

Other associations –

- Pentalogy of Fallot – TOF + ASD/PDA
- Lutambacher's syndrome – ASD + rheumatic MS
- Triology of Fallot – PS + RVH + ASD
- Ebstein's anomaly (PFO/ASD in ≥80%)
- CoA

PFO: In course of time, fibrous tissue develops and permanent anatomical closure occurs by about first year of life. In some cases (20%), the closure remains incomplete known as patent foramen ovale (probe patent).

(**Failure of apposition** of septum primum & septum secundum creating an oblique foramen) in contrast, **in ASD, tissue breakdown** in the middle of septum secundum creating an opening.
(Wadud sir)

Stretched foramen ovale: In later years of life when the patent foramen is stretched for some reasons, it is known as a **stretched foramen ovale (acquired ASD)** and functions like small ASD secundum.

Q. General management of ASD –

- Diuretics
- β -blockers to prevent atrial tachyarrhythmias
- IE prophylaxis if –
 - ASD primum + cleft MV
 - ASD secundum + MVD

Q. Echo findings in ASD –

- **2D:** RA, RV, PA – dilated; paradoxical movement of IVS
- **Subcostal view:** RA, RV – dilated; Echo dropout at the level of IAS
- **Features of pulmonary hypertension in M-mode:**
loss of 'a' dip, systolic notch, reduced EF slope
- **CWD:** Continuous flow from LA to RA through septal defect during both phases of cardiac cycle
- Also measure **PPG across the TV** to see the features of pulmonary hypertension
- **Colour Doppler in Subcostal & A4CH view:** Flow from LA to RA to RV during diastole giving rise to serpentine appearance or butterfly appearance
- Look for associations TAPVC, PAPVC, MVP. (MVP in ASD secundum)
- **TOE:**
- **Contrast echo:** (To see small ASD/PFO, sinus venosus ASD) (Gaffar sir)
 - A small bolus of agitated saline with air bubbles is injected into a peripheral vein.
 - The air bubbles are seen in the RA (RA becomes opacified) and they normally enter into RV.
 - The subject is asked to perform a Valsalva manoeuvre (to increase intrathoracic pressure) when the **air bubbles are seen shunting from the RA to LA** across the ASD.
(POSITIVE CONTRAST EFFECT) Positive Jet
 - A **NEGATIVE CONTRAST EFFECT** is observed when there is an **area of non-contrast in the RA, due to washout effect by normal blood from the LA.**
(LA to RA) Negative Jet

(Black column in contrast)

(In sinus venosus ASD, black column from roof) (Gaffar sir)

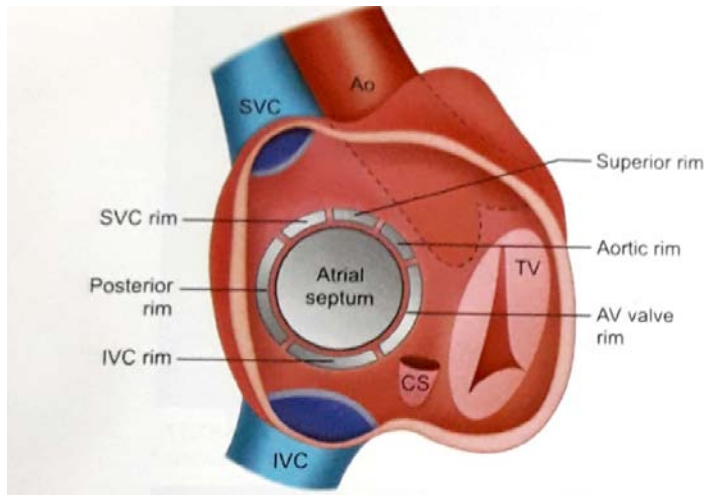
ASD rims/ margins for device closure: (Textbook of echo - Amuthan, Parashar) p.493

Margins of secundum ASD: With advent of interventional closure of secundum ASDs with self-centering double disk nitinol occluder devices, it becomes mandatory to study the relation of occluder device to the neighboring structures.

5 rims/ margins: (Wadud sir says 6 rims)

- **Superior margin**, in relation to SVC (Bicaval view)
(SVC rim= SUPERIOR RIM)
- **Antero-superior margin/ retro-aortic margin**, in relation to ascending aorta & aortic root
(Not mandatory for device closure) PSAX
- **Antero-inferior margin/ mitral margin**, in relation to A-V valves
- **Inferior margin (IVC rim)**, in relation to IVC (Bicaval view)

- **Posterior margin**, in relation to pulmonary veins
(Adequate rim ≥ 5 mm in mandatory)



Class I indications of ASD closure:

- Adequate rim of tissue (>5 mm) from the defects to surrounding structures such as coronary sinus, SVC, IVC, AV valves as well as pulmonary veins
- ASD minimum diameter > 5 mm and <40 mm on echo
- RA, RV enlargement +/- symptoms

Complications of device closure: (Wadud sir)

- CHB
- Failure to implant properly/ improper implantation
- Embolization
- Perforation
- Arrhythmias
- Para-device leakage
- IE

Assessment of ASD for device closure: (Aziz bhai)

- Isolated ASD secundum
- Size <35 mm at rest, <40 mm on stretching
- Adequate (> 5 mm) rim/ margins of ASD, regular rim
- Absence of Eisenmenger's syndrome
- Normal pulmonary vasculature; Qp:Qs >1.5 , PVR <7 Woods Unit (Gaffar sir)
- No other congenital anomaly

Q. If device closure done for ASD with Eisenmenger's syndrome – (Wadud sir)

- Right side – volume overload, RHF
- Left side – inadequate CO

Q. If ASD diagnosed in elderly – (Gaffar sir)

- Theoretically, at any age, ASD should be closed.
- At age 60, can be followed up if asymptomatic. But risk of atrial tachyarrhythmia.
(In neonate, 2 years observation for closure.)

Q. Device closure/ occluder devices done for – ASD/PFO, VSD, PDA (Wadud sir)

- Amplatzer septal occluder
- Figulla Flex II ASD occluder
- STARFlex/ CardioSEAL occluder

Q. Why cardiac cath in ASD – (Trajectory, Oximetry, Pressure, Graphy)

- Only venous access (Both arterial & venous access in other cases) (Gaffar sir)
- **Catheter trajectory:** Catheter pass from RA to LA, 25% on PFO
- **Oximetry:** From oximetry, calculate Qp/Qs ratio
 - Step-up in SVC → ASD Sinus venosus
 - Step-up in high RA → ASD Sinus venosus
 - **Step-up in mid RA → ASD secundum**
 - Step-up in low RA → ASD primum
- **Pressure:** If pulmonary hypertension →
 - **Reversibility test:** 100% oxygen, IV adenosine, GTN
- Qp:Qs ratio >1.5, device closure can be done. PVR <7 Woods unit
- Other associations – TAPVC (TAPVD), PAPVC (PAPVD)

Graphy: Give dye into LA→RA (diagnosis of ASD)

Q. Causes of Oxygen step-up in RA during oxymetry – (Gaffar sir)

- ASD (from LA) (O₂ sat in LA - 95%)
- PAPVC/ PAPVD), TAPVC/ TAPVD (from PV)
- Ruptured SOV (sinus of Valsalva) into RA (from aorta to RA shunt)
- Gerbode defect – LV to RA shunt through atrioventricular septum (from LV)
- VSD with TR (from LV→RV)
- Rarely coronary fistula to RA

Q. Oxygen step-up in oxymetry procedure – (Cardiac cath) (CM Shaheen Kabir p.32)

- Left to right shunt
- Step-up or increase in oxygen saturation in one chamber exceeds the oxygen saturation of a proximal compartment by **more than 7% in case of an atrial shunt** or **more than 5% in ventricular or great vessel shunts**

(Gaffar sir)

Shunt	Target chamber	Previous chamber	Step-up O ₂ sat.
ASD	RA	SVC, IVC	7%
VSD	RV	RA	5%
PDA	PA	RV	3%

In TOF, oxymetry is less important.

Q. Platypnoea-orthodeoxia (P-O) syndrome –

- Dyspnoea and arterial desaturation in upright position and improvement when lying down
- **Causes:** ASD, PFO, Fenestrated IAS aneurysm, Hepatopulmonary syndrome
- Mechanism:

- Under normal condition, shunt from LA to RA in ASD/PFO
- P-O syndrome can be explained on the basis of positional modification of abnormal shunting
- Standing upright could **stretch** the interatrial communication, thus allowing more streaming of venous blood from IVC through the defect
- A persistent Eustachian valve can cause RA to LA shunt despite normal RA pressure.
- Bendopnoea – Dyspnoea on bending forward (like knotting shoe lace). (Gaffar sir)
It means HF control is still inadequate. Also common in COPD.

Q. Classify VSD (Types of VSD) –

According to haemodynamic consequence –	
Restrictive	● Significant pressure gradient between LV & RV; ● Small shunt (Qp:Qs ≤ 1.4:1)
Moderately restrictive	● Intermediate interventricular gradient; ● Moderate shunt (Qp:Qs = 1.4 to 2.2 :1)
Non-restrictive	● Usually > 1 cm² ● Equalization of pressure between ventricles ● Large shunt (Qp:Qs = >2.2 :1)

Anatomically – (Griffin 5e, p.445)

- Membranous – most common, 70-80% of VSDs
- Muscular: 5-20%
- Inlet or A-V canal types: 5-8%
- **Supracristal or subaortic**: 5-7%
 - Located immediately beneath the PV & AV. Often small. Because of their proximity to the AV, aortic leaflet tissue can prolapse through the defect resulting in AR.

Modified Soto's classification – (Satpathy, p.99)

- **Perimembranous VSD** (**Infracristal**, **subaortic**, or membranous): 80%
- **Outlet VSD** (doubly committed, juxta arterial defect, **supracristal**, **subpulmonic**, infundibular or conal): 20-25%
- **Inlet VSD** (non peri-membranous, juxta tricuspid): 5-8%
- **Muscular VSD** (Central, apical, marginal, multiple)

Q. Echo of VSD: (Nanda)

- **Membranous VSD**: seen in PLAX view and 10 o'clock position in relation to AV in PSAX view
- **Supracristal VSD**: 2 o'clock position in relation to AV in PSAX view

Q. Causes of VSD (Ventricular Septal Defects) – (Gaffar sir)

- Congenital – including Gerbode defect
- VSR due to acute MI
 - In apical part in anterior MI
 - In basal part in inferior MI
- Blunt trauma to chest wall
- Acute IE
- Acute rheumatic carditis
- Cardiac surgery

(VSD – left sided murmur)

Q. Swiss cheese VSD – (Gaffar sir)

- Multiple small VSDs in muscular septum (sieve-like fenestration)
- The Swiss-cheese VSD's although small in size **do not close**. (But single muscular VSD can close.)
- The amount of left to right shunt in Swiss cheese defect is small, so there is **no hemodynamic burden**.

Q. Gasul phenomena in VSD – (Satpathy, p.106)

In moderate to large VSD, rarely pulmonary flow may decrease giving rise to symptomatic improvement secondary to acquired stenosis of RV infundibulum. (Functional PS, protective mechanism)

Q. If murmur of VSD disappears, possibilities – (Gaffar sir)

- Spontaneous VSD closure
- Eisenmenger's syndrome

Q. Natural history of Persistent ductus arteriosus (PDA) murmur:

- **Start with silence, end with silence** (Gaffar sir)
- Up to 3 months - **Silent** due to high pulmonary pressure (**Doppler PDA**)
- 3 month to 3 years – **only systolic murmur** (decreased pulmonary pressure)
(Commonly ejection systolic murmur is audible; the diastolic portion of murmur is not appreciable or very short.)
- From 3 years onwards – **Continuous murmur** (PA pressure 30/10-15)
- As pulmonary hypertension develops,
 - S2 becomes prominent with P2 louder than A2,
 - Hyperdynamic precordium becomes quiet,
 - Left parasternal heave felt and LV apex becomes RV type.
 - Cyanosis appears in lower limb (differential cyanosis).
 - The diastolic part of continuous murmur disappears.
 - The long systolic murmur becomes a **short ejection systolic murmur**.
- Eisenmenger's syndrome – **Silent**
 - Florid signs of severe pulmonary hypertension with shunt reversal
 - EDM of PR, PSM of TR

Q. Signs of large PDA – (Satpathy p.118)

- **High volume collapsing** (water-hammer/ bounding) **pulse**, wide pulse pressure
- JVP –
- Hopping carotid pulsation (Satpathy)
- Apex beat – shifted down & out, forceful/thrusting ill-sustained, Hyperdynamic precordium with **forceful** LV apex (Satpathy),
(Volume overload – thrusting unless systolic dysfunction)
- A **continuous thrill** over left second space and sometimes left infraclavicular area up to axillary region may be palpable in children
(**Only systolic thrill** in infants)
- S1 normal, **LV S3** (Volume overload)
- **Continuous murmur**/ Gibson's murmur/ machinery murmur/ (cartwheel, humming top, churning, mill wheel and **train in tunnel murmur**)

(high-pitched, harsh or rasping murmur, better heard in supine position with crescendo & decrescendo quality, increased during inspiration due to fall in PVR), diastolic part of the murmur is usually short, not as long as small PDA.

- Eddies sounds (multiple sound) heard within the continuous murmur, produced by turbulent flow inside the duct

Q.

Silent PDA (Doppler PDA)

Small PDA (Qp:Qs <1.5:1): continuous murmur common.

Moderate PDA (Qp:Qs of 1.5-2.2:1): continuous murmur common.

Large PDA (Qp:Qs >2.2:1): continuous murmur present.

Eisenmenger syndrome: continuous murmur absent; substantial PH, differential hypoxaemia and differential cyanosis.

Small PDA complication: Infective end-arteritis.

Large PDA complication: Early PH & shunt reversal, LVF. (Gaffar sir)

Doppler PDA – Sometimes a small duct, which is narrow but have a patent lumen may not produce audible murmur, it is only detected by colour Doppler echocardiogram. (Satpathy)

Q. Continuous murmur - D/D (Raghawa Rao)

Continuous murmur: begins in systole & continues without interruption through S2 into all or part of the diastole without change in the character of the murmur.

(starts in late systole, obscuring S2, till early diastole) (Gaffar sir)

- PDA (Gibson murmur)
- Aorto-pulmonary window
- Rupture sinus of Valsalva (RSOV)
- (Cervical) Venous hum
- Pulmonary A-V fistula
- Coronary A-V fistula (Coronary cameral fistula)
- CoA with periscapular collaterals
- Mammary soufflé (of pregnancy, 2nd & 3rd trimesters & early postpartum)

Continuous murmur should be differentiated from –

- VSD + AR (Perimembranous VSD, RCC prolapse) (Gaffar sir)
- AS + AR
- MS + MR

PDA:

- best heard in left infraclavicular & pulmonary area during expiration,
- Crescendo-decrescendo murmur, peaks at S2, associated with thrill

RSOV: best heard at lower left sterna border, H/O sudden starin/ weight-lifting

Venous hum: (due to ↑velocity of blood flow)

- In young, lean & thin person,
- Hyperdynamic circulation,
- Heard in sitting posture, (↓ in supine)

- Obscured by digital compression at root of the neck (IJV)

Q. Auscultation of PDA –

P₂ – loud if PH

Splitting narrow

S₃ may be audible (Volume overload)

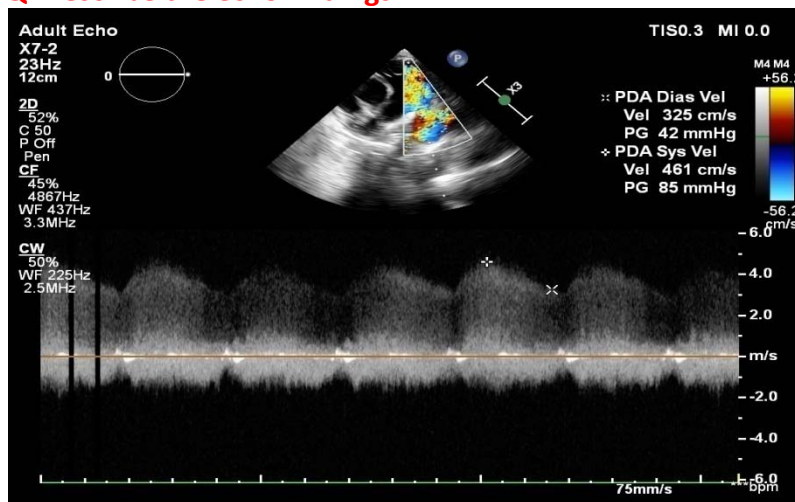
Apical diastolic murmur without thrill (Functional MS in large PDA)

Q. Echo of PDA –

On echo of a children, LV dilated – Suspect PDA (Gaffar sir)

- 2D – LA, LV dilated (LV volume overload), LA:Ao >1.3 (Luthra p.149)
- Doppler echo – (Wadud sir)
 - o Colour flow mapping – PSAX – at the level of AV -
Retrograde **mosaic jet of flow** (high velocity jet directed back toward the probe and PV) from LPA to dilated MPA (Luthra p.149)
 - o PSAX – CW – Classic high velocity continuous flow (**Picket-fence appearance**), allows for measurement of peak velocity (Washington Manual, p.251)

Q. Describe the echo findings –



2D-guided colour-flow mapping & spectral Doppler in PSAX view – sample volume placed at bifurcation of MPA showing – Continuous spectral signal above the baseline giving picket-fence appearance (Aziz Bhai)

Q. Cardiac cath in PDA

- Right heart – Cournand catheter (end hole) – to see **trajectory (right to left)**
Inverted hair pin appearance crossing the diaphragm
- Left heart – Pigtail catheter (both end & side hole) – **Arch aortography** –
Dye will come to lung (PDA confirmed)
- Reversal – PA dye will go to descending aorta (Nazrul sir)
- Pressure study – (Nazrul sir)
 - o Normal if no pulmonary hypertension
 - o If pulmonary hypertension: RA, RV, PA – dilated
 - o If Eisenmenger's syndrome: PA = Ao = RV = LV

Q. Interpret data with possible diagnosis.

Saturation data	Pressure data	
SVC - 60, IVC - 65,	RA - 5,	Qp 9.5 L/min
MVO2 - 61.25,	RV - 64/6 (25),	Qs 3.1 L/min
RA - 60 , RV - 72 ,	RV - 64/23 (39),	L-R shunt ventricular 0.9L/min
PA - 88, PV - 98,	LV - 104/10, Aorta - 107/46(73)	Aorta to pulmonary 5.5 L/min
Aorta - 98,	LA - 8	

- VSD with PDA with PH.

Q. ASD causes Right heart failure

VSD, PDA – Left heart failure

PDA – LV enlargement

ASD – RVH

VSD – initially LVH, followed by RVH

Q. Coarctation of aorta (CoA) – (Hurst p.1384, Braunwald p.1559)

- 7% of CHD, M>F (1.5:1) (2-5 times more common in male- Braunwald's), 2:1 (Griffin)
- Diffuse arteriopathy, cystic changes in aortic media
- **Associations:**
 - o BAV – 50% (Hurst), 50-85% (Griffin)
 - o Intracranial aneurysms, often in the circle of Willis – upto 10% (Hurst), (2-10%) (Braunwald), 3-5% (Griffin)
 - o Turner's syndrome (35% have CoA)
 - o VSD, PDA
 - o Congenital malformations of MV (smaller orifice, supralvalvular ring, parachute MV resulting from single PM), MR
 - o Valvular & subvalvular AS
- **Shone complex:** Multiple left-sided heart lesions may be associated with CoA
- **Systemic arterial hypertension in upper extremities** (differential SBP at least 10 mmHg) (Brachial artery pressure > Popliteal artery pressure) (A normal person should have a 5-10 mmHg increase in SBP in the lower extremities compared with upper extremities. Absence of this increase or presence of a decrease in the lower extremities should arouse the suspicion of CoA.)
- **C/C:** Headache, nosebleeds, cool extremities, leg weakness/ Claudication with exertion; More serious manifestations - Angina, HF
- Radio-femoral delay is evident unless significant AR.
- **Auscultation:**
 - o **Interscapular systolic murmur** emanating from the coarctation site
 - o Widespread crescendo-decrescendo **systolic murmur** throughout the chest wall from the intercostal collateral arteries
- Fundus: 'Cork-screw' tortuosity of retinal arteries
- **Cause of death:**
 - o CHF (usually in >30 yrs)
 - o Aortic dissection or rupture
 - o Complications of infective end-arteritis/ IE
 - o ICH from Berry aneurysms
 - o CAD

- Concomitant AV disease
- **Significant CoA:** A gradient of >20 mmHg across the coarctation site at angiography with or without proximal systemic hypertension. (A pullback >20mmHg signifies haemodynamic significance)
- **Management:**
 - Surgical:
 - Excision of the narrowed segment and end-to-end anastomosis of the para-coarctation aorta – preferred method of initial repair
 - Prosthetic overlay grafts –
 - Subclavian flap repair
 - Subclavian patch aortoplasty
 - Interposition graft – excision of the narrowed segment & placement of a prosthetic conduit
 - Prosthetic conduit placement from ascending aorta to descending aorta – in patients with complete interruption
 - Intervention: Percutaneous balloon angiography with stenting

Q. Holt-Oram syndrome –

- An autosomal dominant syndrome
- Radial abnormalities of the forearm and hand
- Associated with **secundum ASD** (most common), VSD or rarely other cardiac malformations
- Phenotypic variability within & between families (mutation in TBX5)

Q. Lipid lowering therapy – (Gaffar sir)

For LDL-C: Statins, Ezetimibe, Resin (Cholestyramine) (Mnemonic-**ESR**)

For TG: Fenofibrate, Nicotinic acid, Fish oils (Omega-3 fatty acid) (Mnemonic-**FNF**)

PCSK-9 (Proproten Convertase Subtilis/Kexin type 9) inhibitors: Alirocumab, Evolocumab

Statins:

1. Atorvastatin 10, 20mg
2. Rosuvastatin 5 (starting dose for Asian), 10, 20mg
3. Fluvastatin 20, 40, 80mg
4. Pitavastatin 1, 2, 4mg
5. Pravastatin 20, 40mg
6. Simvastatin 20, 40mg
7. Lovastatin 20mg

Pleiotropic effects of statin therapy: (Wadud sir) [Pleiotropic = multiple]

1. **Plaque stabilization** (Very important)
2. Anti-platelet effect (Stabilize platelet), Anti-thrombotic (↓ fibrinogen) (Opie p.430)
3. Anti-inflammatory effect (Opie p.430)
4. Immunomodulatory effect
5. Reduce endothelial dysfunction (by NO production)
6. Anti-oxidant (Opie p.430)
7. Vascular cytoprotection,
8. Angiogenesis
9. Reduce cardiovascular mortality & all cause mortality
10. Regression of coronary atherosclerosis

11. Decrease proteinuria & progression of CKD

(Beneficial effect on disease states unrelated to atherosclerosis such as arrhythmias, cancer, Alzheimer's disease and other neurodegenerative disorders) (Opie p.430)

Pregnancy warning (Opie)

- Statins must not be prescribed to women who are pregnant or who are planning to become pregnant because cholesterol is essential to foetal development.
- Statins are excreted in the mother's milk, so women taking statin should not breastfeed.
- Women desiring to become pregnant should stop statins for approximately 6 months before conception.
- If a patient becomes pregnant when taking statin, therapy should be discontinued and the patient appraised of the potential hazards to the foetus.
- Only dietary modification for them.
- Young obese male on statin planning for baby – Give statin carefully (as main nutrition of sperm is lipid) (Wadud sir)

Side effects of statin therapy: (Opie)

- Liver damage
- Myopathy (Muscle pain, rhabdomyolysis, AKI due to myoglobinuria)
- New diabetes
- Cognitive side effects (memory loss, confusion)

FDA stated that it believes that increased risks for new diabetes & cognitive problems are small and outweighed by the cardiovascular benefits of statin therapy.

Q. Statin therapy – (Loading dose 80mg atorvastatin) (AHA % reduction; ESC set target)

- **High-Intensity statin therapy:**
 - Daily dose lowers LDL-C, on average, by approximately $\geq 50\%$
 - Atorvastatin 40-80 mg, Rosuvastatin 20-40 mg
- **Moderate-Intensity statin therapy:**
 - Daily dose lowers LDL-C, on average, by approximately 30% to <50%
 - Atorvastatin 10-20 mg, Rosuvastatin 5-10 mg
 - Fluvastatin 40 mg BID or 80 mg XL daily
 - Pitavastatin 2-4 mg
- **Low-Intensity statin therapy:**
 - Daily dose lowers LDL-C, on average, by <30%
 - Simvastatin 10 mg, Pravastatin 10-20 mg
 - Fluvastatin 20-40 mg
 - Pitavastatin 1 mg

Q. Lipid profile – Fasting is not necessary unless TG >400mg/dL (Wadud sir)

- Friedewald formula:
 - $LDL-C = TC - HDL - TG/5$ (mg/dL)
 - $LDL-C = TC - HDL - TG/2.2$ (mmol/L)
 - This formula is unreliable when
 - TG >400mg/dL (4.5 mmol/L) (ESC 2011)
 - TG >350 mg/dL (4.0 mmol/L) (Davidson)

o Alternatively, **Apo B, Apo B/Apo A1 ratio**

	Reference range (Lalpath)	
Apolipoprotein (Apo A1)	105-175 mg/dL	
Apolipoprotein (Apo B)	60-135 mg/dL	>135 Hyperapobetalipoproteinaemia, ↑CAD risk
Apo B/ Apo A1 ratio	0.35-0.98	>0.98, ↑CAD risk

- TC (Total cholesterol) = LDL-C + HDL-C + VLDL
- VLDL = TG/5 (mg/dL) = TG/2.2 (mmol/L)
- Lipid conversion factor, **mg/dL to mmol/L**
 - o For TC, HDL-C, LDL-C, VLDL and non-HDL-C: **divide mg/dL by 38.67**
 - o For TG: **divide mg/dL by 88.57**
- TC (Total cholesterol) conversion (equivalent) (ESC 2011)

mmol/L	mg/dL
4 ≈	150
5 ≈	190
6 ≈	230
7 ≈	270
8 ≈	310

- **Bad cholesterol:** LDL-C (Cholesterol>TG), VLDL (TG>Cholesterol)

Q. If patient is on treatment for infertility, withhold statin – (Wadud sir)

- As it can decrease sperm motility
- Dietary modification
- Omega-3 fatty acid can be given

Q. Statin-induced myopathy –

- Dose-dependant
- Start with low dose in hypothyroidism, hypokalaemia
- Role of Coenzyme Q in statin-induced myopathy: No RCT

Q. Discussion in Statin therapy CME –

- Resistant dyslipidaemia – LDL-C >300 [found in FH(familial hypercholesterolaemia)]
- Hyper HDL-C can ↑CAD
- LDL-C can be lowered up to 45
- **Targets:** Total-C <150, TG <150, HDL-C >40
LDL-C <70 (High risk), <100 (Moderate risk), <130 (if no risk factor)
- SGPT ↑2.5 fold→↓dose; ↑3 fold→↓dose by half ; ↑5 fold→ Stop;
- LDL aphaeresis can be done in resistant hypercholesterolaemia due to FH
- HDL-C ↑ by which statin – Rosuvastatin, Atorvastatin >20mg/day (along with ↓LDL-C)
- Fenofibrate ↑gall stones; Statins ↓ gall stone (Wadud sir)
- Atorvastatin before meal, Rosuvastatin meal independent

Q. β-blocker in Heart failure –

1. Carvedilol (**Rule of 2**) (½+0+½, ↑2 weekly, upto target dose 25 bd or 50 mg daily) (Wadud sir)
2. **Metoprolol succinate** (Betacloc XR 50, 100)
3. Bisoprolol

4. Nebivolol

(Metoprolol tartrate is available as Betaloc/ Presonil/ Selomet)

Carvedilol in HF – (Khaleq sir)

- Block sympathetic drive
- Peripheral vasodilatation
- Anti-oxidant

Q. Spironolactone in HF – (Khaleq sir)

- Anti-aldosterone
- Diuresis
- Decrease myocardial fibrosis (reduce mortality)
- Decrease myocardial arrhythmia

(Keep K⁺ in upper normal limit in ischaemia/ HF settings)

Q. β -blocker in acute MI – (Khaleq sir)

- IV β -blocker reduce cardiac rupture
- Decrease rate and myocardial oxygen demand
- Anti-arrhythmic
- Reduce infarct size

Q. Ramipril – Target dose 20 mg daily (Wadud sir)

Q. Starting dose of α -blocker (Prazosin) – initially 0.5 mg bd

Q. Vasodilator β -blocker – can be used in Peripheral arterial disease –

- Carvedilol
- Labetalol
- Nebivolol

Q. Rate limiting beta blocker –

Propranolol, Bisoprolol, Metoprolol, Sotalol

Q. Nebivolol –

Less erectile dysfunction

40% renal & 60 faecal (biliary) excretion

Q. β -blockers in renal impairment – (only liver metabolism)

MCP

- Metoprolol, Carvedilol, Propranolol

Q. Renal-liver metabolism (50%-50%) β -blockers

- Bisoprolol, Nebivolol, Labetalol

Q. 100% renal metabolism β -blockers

- Atenolol (Betanol, Cardipro, Tenoloc, Tenoren),
- Sotalol [Tab. Sotalax 80mg (Unimed)]
- Acebutolol

Q. Cardiac Drugs causing flare of psoriasis –

- **β-blockers**
- **ACEi/ARB** (Antimalarials, Lithium, NSAIDs, Anti-TNF- Infliximab)

(Other exacerbating factors – Trauma, infections, drugs, emotions, stress, sunlight) ‘TIDES’

Q. Alcoholic patient develops palpitation –

AF (Holiday heart syndrome/ Saturday night syndrome)

Q. Causes of variable S1 –

1. **AF,**
2. **CHB (A-V dissociation)** (In CHB – Irregular Cannon wave + variable S1)
3. Mobitz type I HB with A-V dissociation
4. VT with A-V dissociation
5. Atrial tachycardia with varying block

Q. CHB vs sinus bradycardia – Clinically in CHB

- Rate 40-50/min
- Variable S1
- Irregular Cannon waves in JVP

Q. In AF – always check heart rate, rather than pulse rate

AF – Pulse deficit >6, PVC – Pulse deficit <6;

Irregularly irregular pulse; **variable** in volume

JVP – Loss of ‘a’ wave, single systolic (v) wave

- **The more the pulse deficit, the more chance of haemodynamic instability.**

Q. Causes of irregularly irregular pulse – (Gaffar sir)

1. **Frequent PVCs/ atrial ectopics**
2. **Atrial fibrillation**
3. Atrial flutter with variable block
4. Atrial tachycardia with variable block
5. **Multifocal atrial tachycardia (MAT)** (rate >100/min)

(Multiformed atrial rhythm/ Wandering atrial pacemaker – rate <100/min)

Q. How will you differentiate clinically irregularly irregular pulse of AF from that of frequent PVC? (Gaffar sir)

	AF	Frequent PVC
Pulse deficit	>6/min (>10)	<6/min (<10)
Apex	Variable S1	
JVP	Loss of ‘a’ wave, Single systolic wave (v)	
Exercise	Do not disappear, May aggravate	May disappear

Q. Causes of regularly irregular pulse –

1. **Sinus arrhythmia**
2. **Pulsus bigemini, trigemini, quadrigemini**
3. Pulsus alternans
4. 2nd degree (**Mobitz type I**) heart blocks, (?)1st degree HB

Q. Criteria of Premature ventricular contraction (PVC) – (Baltazar)

- 1) Not preceded by P waves
- 2) Wide ($\geq 0.12s$) QRS complex
- 3) ST segment and T waves are in opposite direction (discordant) to the QRS complex
- 4) QRS complex is followed by a pause that is usually fully compensatory

Classify PVC –

- Unifocal –
 - o originate from a single location in the ventricle,
 - o uniform & identical configuration
- Multifocal –
 - o originate from two or more locations in the ventricle
 - o different configuration
- PVCs in bigeminy
- PVCs in trigeminy
- PVCs in quadrigeminy
- Paired PVCs: in pairs or in couplets when two PVCs occur consecutively

Interpolated PVC:

- The PVC is interpolated when it is inserted between two sinus impulses without altering the basic sinus rate
- Not followed by a pause

Coupling interval:

- Distance between the PVC and the preceding QRS complex.
- Coupling interval of most PVCs is **usually constant**.
- PVCs manifesting the **R on T phenomenon** have short coupling intervals, usually measuring about ≤ 0.40 seconds. The PVCs occur at the downslope of the T wave of the previous complex, which corresponds to the vulnerable period of the ventricles. This may potentially trigger a ventricular arrhythmia.
- **End-diastolic PVCs** have long coupling intervals because they occur very late during diastole after the sinus P is inscribed.

Ventricular parasystole:

A PVC is considered parasystolic when all of the following features are present.

- Variable coupling intervals.
- Fusion complex.
- Constant intervals between the ectopic ventricular complexes.
(The ventricular complexes are mathematically related.)

Localizing the origin of PVCs:

- LV origin – RBBB pattern on ECG

- RV origin – LBBB pattern on ECG

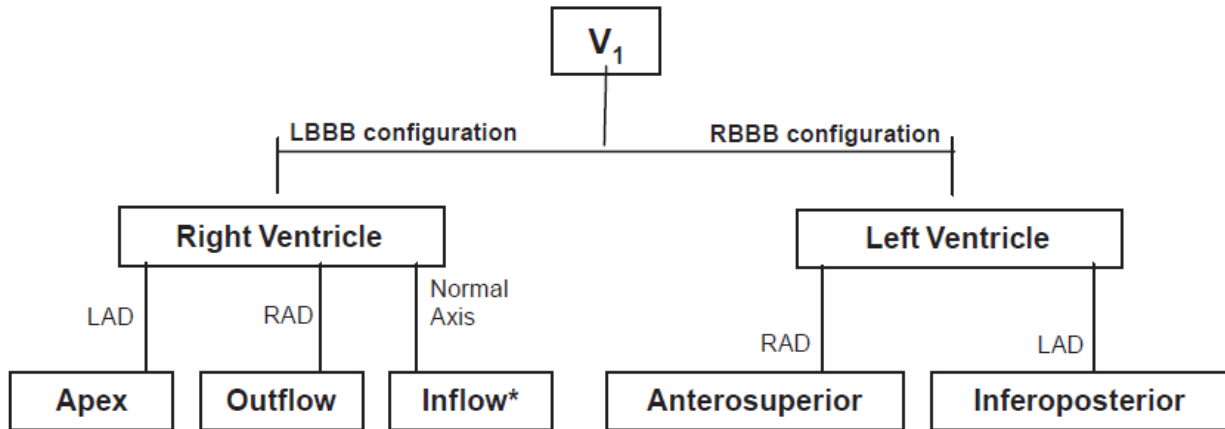


Figure 21.13: Localizing the Origin of the Premature Ventricular Complex (PVC). See text for explanation. LAD, left axis deviation; LBBB, left bundle branch block; RAD, right axis deviation; RBBB, right bundle branch block. *When the PVC has a normal axis, the PVC could originate from the right ventricular inflow or the area between the right ventricular apex and right ventricular outflow.

Rx of PVC –

- β -blockers (Do not give in asthma)
- rate-limiting CCB (if pulse & BP allow)
- Amiodarone (if above two cannot be given)

(Patients who have >6 PVCs/hour have 60% relative ↑risk of SCD.) (Griffin 5e, p.68)

Q. Premature supraventricular complex –

- **Premature atrial complex (PAC)** (Baltazar p.172)
 - P wave premature, before QRS
 - P wave different morphology, may be difficult to recognize if hidden in the T wave
 - PR normal or prolonged
 - P wave may be blocked & not followed by QRS (only a pause)
 - QRS usually narrow, may be wide if conducted aberrantly or pre-existing BBB
 - Pause after PAC – not fully compensatory
- Blocked PAC – mistaken for Sinus arrest or SA block, second-degree HB (Blocked PAC should always be suspected before sinus node dysfunction is considered)
- Blocked PAC in bigeminy – mistaken for sinus bradycardia
- PAC conducted with aberration (wide QRS) – frequently confuses with PVC
- **Premature junctional complex (PJC)** – originating from AV node or bundle of His
 - Retrograde P wave before QRS complex
 - Retrograde P wave after QRS complex
 - Retrograde P wave synchronous with QRS complex
- AV node –
 - Upper AV node (AN, atrionodal region) – capable of firing spontaneously
 - Middle AV node (N, nodal region) – NOT capable of firing spontaneously

- o Lower AV node (NH, nodo-His region) – usually the site of origin of junctional impulse

Q. Mobitz type I Heart blocks – block in AV node (Gaffar sir)

Q. Mobitz type II Heart blocks – (Intra- or infra-Hisial)

Q. Supraventricular tachycardia (SVT) – Presentations

- Sudden palpitation, chest pain, dyspnoea
- Polyuria,
- Haemodynamic instability, hypotension → Lightheadedness, dizziness
- ECG – 4 features
 - o Heart rate – 150-250/min
 - o Narrow complex tachycardia
 - o Rhythm – regular
 - o P wave – absent

Q. SVT – Non-pharmacological manoeuvres (act by ↑vagal tone)

1. Carotid sinus massage (see carotid bruit first), in any one side, firmly
2. Valsalva manoeuvre (forced expiration against close glottis)
3. Immersion of face in cold water (like **duck diving**)
4. Eyeball compression
5. Deep pain stimulation

Q. Drug Rx in SVT –

- IV Adenosine with normal saline flush (exclude bronchospasm)
 - o (**Rule of 2** – over 2 sec, after **2 min** – double the dose)
 - o (6 – 12 – 24 mg)(1 – 2 – 4amp) (**t½ of adenosine 6-10s**) (Gaffar sir)
 - o (6 – 12 – 12 mg), repeat the cycle (Wadud sir)
- IV Verapamil (5-10 mg slow IV) (mix with NS) (ABM Abdullah ECG - 10mg)
- IV Amiodarone (with 5% D/A) (if mix with NS → Na-isodide)
- Digoxin – oral, IV, IM (if above drugs are not available) (Gaffar sir)
- If Haemodynamically unstable – DC shock (Indications below)
- Prophylactic oral therapy in frequent or disabling SVT – β-blocker, verapamil, disopyramide, digoxin
- In WPW syndrome – Transvenous RFA
- Anti-tachycardia pacing (Overdrive atrial pacing)

(Verapamil should be avoided if QRS >0.12s or **H/O WPW syndrome** or on β-blocker)

Q. Causes of SVT –

- Physiological – anxiety, tension, tea, coffee, alcohol
- Pathological –
 - o IHD including AMI,
 - o Thyrotoxicosis,
 - o WPW syndrome,
 - o Digitalis toxicity,
 - o Electrolyte imbalance (Hypokalaemia) (Gaffar sir)

Q. Indications of DC shock in SVT –

- Haemodynamically unstable (HR >200/min, SBP <90 mmHg)
- Intractable chest pain
- Heart failure
- Unconsciousness/ Impaired consciousness

Q. Investigations in SVT to find out cause–

- ECG after cardioversion – WPW syndrome
- Troponin I (AMI)
- **Serum electrolytes** (Hypokalaemia) (Gaffar sir)
- Echocardiography
- Thyroid function tests
- CBC (Hb for anaemia, TC for infection) (Gaffar sir)
- Chest X-ray P/A view (Features of MS, infection)
- CAG (in recurrent SVT, after 35 years of age) (Gaffar sir)
- EPS (Aberrant pathway)

Q. Long term management of SVT –

- Structurally normal heart with infrequent event –
 - Reassurance,
 - Avoidance of precipitating factors,
 - Physical manoeuvres
 - Low dose β -blockers/ verapamil
- Structurally abnormal heart with frequent episodes, not responding to medications – (EPS + RFA)

Q. STV with asthma, hypotension, thyroid abnormality – choice of drug

- Asthma – No adenosine,
- Hypotension – No verapamil,
- Thyroid abnormality – No Amiodarone.

Digoxin can be given.

Q. Frank-Starling law –

Within physiological limit, force of contraction is proportional to initial length of cardiac muscle fibre.

Q. Cardiac arrest – Types

Shockable	Non-shockable
Pulseless VT	Asystole
Pulseless electrical activity (PEA)/ Electromechanical dissociation (EMD)	
VF	

Q. What is EMD/ PEA – (Gaffar sir)

- No cardiac mechanical activity – No pulse, No BP, No heart sounds
- But bizarre electrical activity in monitor
- Suspect Free wall rupture, pericardial tamponade
- Almost 100% mortality

Q. Types of VT –

- Non-sustained (<30s) – salvo of ≥ 3 consecutive PVC at a rate of $>100/\text{min}$, terminating in < 30 seconds. Detected by ECG, Holter or bedside **monitor**
- Sustained ($>30\text{s}$) – VT lasting $>30\text{s}$, or requiring termination due to haemodynamic compromise in $<30\text{s}$
- Short runs of VT – detected by ECG, Holter, monitor
- **Fascicular VT** – uncommon WCT with **characteristic RBBB pattern** (not very wide RBBB), usually with LAD and a superior axis – positive in aVR. It is only WCT in which **verapamil** can be used & most effective drug.
- **RVOT VT** – **Characteristic LBBB pattern** with an inferior (vertical) axis.

According to haemodynamic status – (Gaffar sir)

1. Haemodynamically stable (VT with normal Pulse+BP)
2. Haemodynamically unstable (VT with hypotension/ shock)
3. **Pulseless VT** (Cardiac arrest - Indication of DC shock)

Causes of VT –

- Acute MI, IHD
- Ventricular aneurysm
- Myocarditis
- Cardiomyopathy

C/F – palpitations, ...

VT and haemodynamic stability – (Gaffar sir)

HR	Haemodynamically unstable
$>200/\text{min}$	100%
150-200/min	70%
100-150/min	30%

Q. Examination findings in VT – (Baltazar) WCT/ BCT

- Irregular Cannon waves in the JVP (Regular Cannon waves only if Ventricular tachycardia (VT) 1:1 retrograde conduction) (Raghawa Rao)
- **S1 – variable intensity**
- Pulse – variable volume

Q. Brugada criteria of VT – (Sensitivity 99% & Specificity 96.5%) (Griffin 5e, p.318)

Step I: **Absence of RS complex in all precordial leads** is VT

Step II: **R to S interval $> 100\text{ ms}$** is VT

Step III: **AV dissociation** is diagnostic of VT (100% specific)
(**More QRS complexes than P waves**)

Step IV: **Morphology criteria in leads V1 and V6** – WCT/ BCT with RBBB/ LBBB morphology

- **RBBB morphology:** monophasic R wave (R, RR', rR', Rr') or biphasic QRS (Rs, qR) in V1 AND any of these QRS pattern (qR, qr, QS, R, R/S ratio <1)
- **LBBB morphology:** R ≥ 40ms, RS ≥ 70 ms, slurred S in downslope AND any q wave (qR, qr, QS, QRS) in V6.

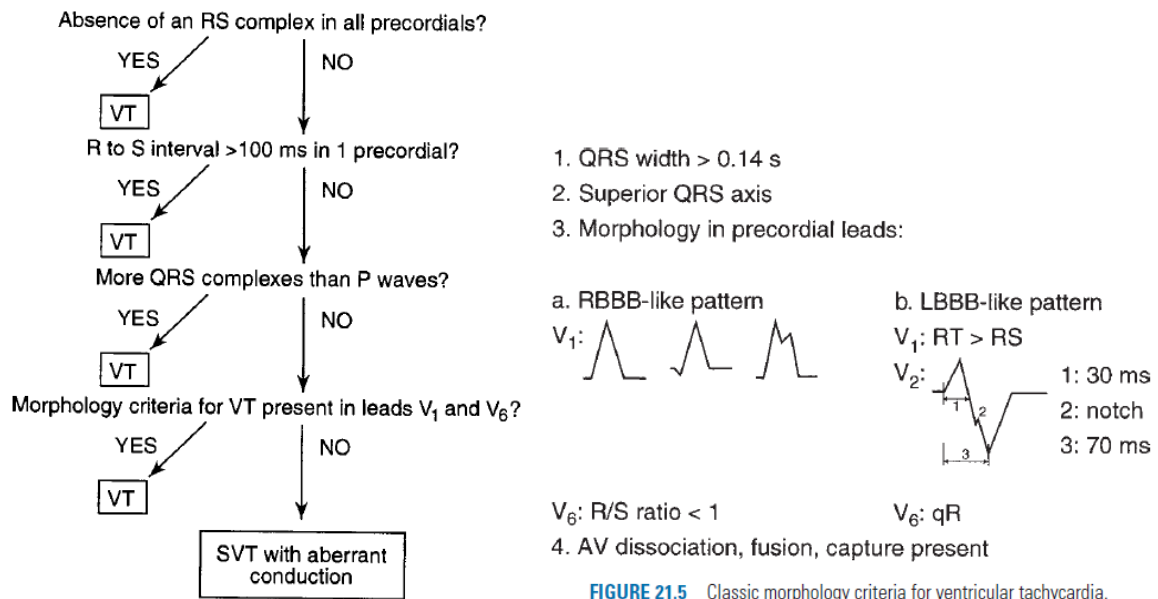


FIGURE 21.5 Classic morphology criteria for ventricular tachycardia.

Q. Ventricular tachycardia (VT) –

- QRS width > 140 ms
- aVR – positive
- may be **slightly irregular** (due to capture beat & fusion beat) (Wadud sir)
(VT is generally regular in rate and appearance, although it can be polymorphic in appearance, slightly irregular with respect to rate, and may have capture and/or fusion beats within it.) (Griffin 5e, p.318)
- Brugada criteria – (if all no → SVT with aberrant conduction)
- RBBB pattern (arises from LV) – left rabbit ear is taller than that of right (V₁) and small r & large S (rS) in V₆ (**Marriott's sign**)
- **Brugada's sign** – The distance from the onset of the QRS complex to the nadir of the S-wave is >100 ms. (LITFL.com)
- **Josephson's sign** – Notching near the nadir of the S-wave. (LITFL.com)

Q. Causes of diastolic dysfunction/ diastolic HF (Gaffar sir)

1. Old age (>50)
2. Female,
3. **Systemic hypertension (Hypertensive heart disease)**, AS, HCM, CoA → LVH
4. **DM,**
5. **IHD,**
6. RCM, Myocardial infiltration/ storage disease
7. Pericardial effusion, cardiac tamponade, CP

Q. Causes of systolic HF – (Gaffar sir)

- IHD
- Myocarditis
- Cardiomyopathy (DCM)

Q. Differentiate systolic heart failure vs Diastolic heart failure –

	Systolic HF	Diastolic HF
Age	Relatively early age	Advanced age/ elderly
Sex	Male > Female	Female > Male
Pulse	Low volume	Normal/ high volume
Systolic BP	Low	Normal/ High
Precordium		
Apex beat	Shifted down & out, Soft , diffuse, Impalpable	Not shifted, Heaving
S1, S2	S1 soft, S2 normal	S1 Normal, A2 may be loud in long-standing hypertension
	S3 gallop (Volume overload)	S4 gallop, (Pressure overload)
Lung	Lung creps	
Echo	Eccentric LVH	Concentric LVH
Treatment	Digoxin may be required	Digoxin is contraindicated

In systolic HF, apex beat may not be shifted due to COPD.

No changes in JVP in Left heart failure unless CCF.

Q. Heart failure according to Ejection fraction – ESC 2016 guidelines

HFrEF (EF <40%)

HFpEF (EF ≥50%)

HFmrEF (HF with mid range EF) – **(LVEF 40-49%)** – most probably have primarily mild systolic dysfunction, but with features of diastolic dysfunction

Q. Diagnostic criteria of HFpEF – (Griffin 5e, p.120)

- 1) LVEF >50%
- 2) Evidence suggesting diastolic dysfunction on diagnostic testing –
 1. LVEDP >16 mmHg or PCWP >12 mmHg
 2. E/e' >15
 3. Lateral e' velocity <10 cm/s or septal e' velocity <8 cm/s
 4. Abnormal mitral inflow Doppler pattern suggesting impaired relaxation (E/A <1), E/A pseudonormalization or restrictive physiology (E/A >2)
 5. Elevated plasma natriuretic peptide levels (NT-pro-BNP >220 pg/ml or BNP >200 pg/ml)

Q. NPs (Natriuretic peptides) / BNP and NT-proBNP –

Q. Causes of right heart failure –

- Intrinsic – RV Infarction
- Outflow obstruction – Acute massive pulmonary embolism
- Extrinsic – Pericardial tamponade, constrictive Pericarditis

(Changes in intrathoracic pressure affect pulsus paradoxus, but doesn't affect in constrictive Pericarditis.)

Q. Right heart failure – Rx with fluid
Left heart failure – Rx with diuretics

Q. Reversible factors in HF –

(Factors that may precipitate or aggravate HF in patients with pre-existing heart diseases – MI or myocardial ischaemia)

1. Intercurrent illness – infections
2. Arrhythmias – AF
3. Inappropriate reduction of therapy/ non-compliance
4. Conditions with increased metabolic demand – anaemia (Check Hb), pregnancy, thyrotoxicosis (Check TFT)
5. IV fluid overload – postoperative iv fluid
6. Pulmonary embolism
7. Drugs with fluid retaining properties – NSAIDs, Steroids,
8. Drugs with negative-inotropic properties – Beta blockers
9. Salt intake
10. Diuretic resistance
11. Other associated conditions related to Rx failure – renal impairment, liver disease, Nephrotic syndrome

(Chronic HF – Address anaemia, thyroid, anxiety)

Q. Ectopic beat & escape beat – (Gaffar sir)

Premature beat – ectopic beat (early), rate high

Delayed beat – Escape beat (compensatory mechanism) late, rate low

β -blocker in premature beat.

No β -blocker in escape rhythm.

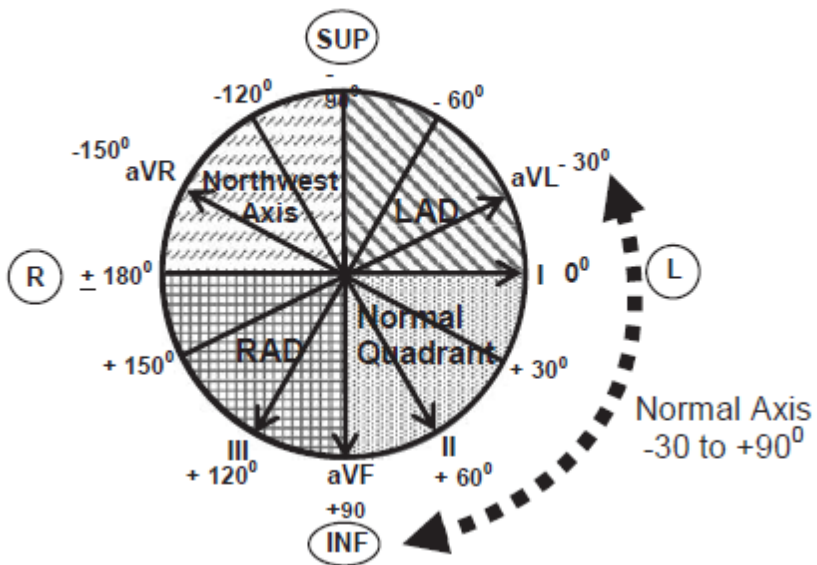
Q. Determination of cardiac axis – (Wadud sir)

- See only lead I and II
- Lead I and II positive: Normal axis
- Lead I positive & lead II negative: LAD
- Lead I negative & lead II positive: RAD

Q. Causes of RAD (right axis deviation) – (Wadud sir)

- 1) RVH (any condition)
- 2) Left posterior hemiblock
- 3) Dextrocardia (true/ technical)
- 4) High-lateral MI

Q. Cardiac axis –



Normal axis: from $+90^{\circ}$ to -30°
 LAD: from -30° to -90°
 RAD: from $+90^{\circ}$ to $+180^{\circ}$
 Northwest axis/ extreme axis: from -90° to $+/-180^{\circ}$
 (Normal axis: -30° to $+110^{\circ}$) (Wadud sir)

Q. Pairs of perpendicular leads –

I, aVF; II, aVL; III, aVR

Q. ECG showing tall R in V_2 –

Exclude True posterior MI.

If patient is young, healthy – possibility of Cardiac rotation counter-clockwise (early transition).
Heart moves to the right, LV moves anteriorly.

Q. Cardiac rotation –

Clockwise (CW) rotation or delayed/late transition:

- When the heart rotates clockwise, the **transition zone**, which is usually V_3 or V_4 , **moves to the left toward V_5 or V_6 .**
- RV moves more anteriorly.**

(When the apex of the heart viewed from under the diaphragm, the front of the heart moves to the left, causing the RV to move more anteriorly).

Counter-clockwise (CCW) rotation or early transition:

- When the heart rotates counter-clockwise, the **transition zone** moves earlier **toward V_1 or V_2 .**
- LV moves more anteriorly.**

(When the apex of the heart viewed from under the diaphragm, the front of the heart moves to the right, causing the LV to move more anteriorly).

Q. Association of WPW syndrome –

- Ebstein's anomaly (always type B, LBBB pattern)

(10-25% have accessory pathway)

- HOCM
- MVP

Frequently there is >1 accessory pathway (e.g. in Ebstein's anomaly) and antidromic tachycardia is more common in patients with multiple pathways. (Swanton)

Q. Ebstein's anomaly –

- Marked apical displacement of septal (STL) & posterior (PTL) leaflet of TV,
- Anterior leaflet is elongated (sail-like) and attached to RV free wall
- Significant TR (at least moderate TR) (TV coaptation is apically displaced)
- Association – ASD/PFO (found in ≥80%)

Echo feature:

- Septal leaflet is attached to IVS, **≥10 mm** inferior to the AML (Luthra)
Apical displacement of TV compared to MV **> 8mm/m²** (Griffin, Washington)
- TR, assess pulmonary pressure
- ASD/PFO (found in ≥80%)

(Echo report –

RA – hugely dilated; RV – small, atrialized; IAS – echo dropout 30 mm,
TV – ATL is long, slender and sail-like; STL & PTL – apically displaced ----mm/m²

Impression:

- Congenital heart disease, Ebstein's anomaly, TV – (),
- Large ASD secundum with short posterior & IVC rims with shunting right to left
- No pulmonary atresia, No VSD or PDA
- TR – Grade II,
- No RWMA, Good LV systolic function (LVEF-____%)

Q. Subclavian steal –

Due to critical stenosis of subclavian artery, proximal to the origin of vertebral artery → during hand exercise, **reverse flow in the ipsilateral vertebral artery** → vertebrobasilar ischaemia → dizziness, vertigo, syncope

Q. Coronary steal –

In a patient with CABG with LIMA/RIMA graft,

Due to critical stenosis of subclavian artery → during hand exercise, **reverse flow in the LIMA/RIMA** → decreased blood flow to heart muscle

Q. Causes of ischaemic chest pain in severe AR –

- Venturi effect, due to **coronary underfilling during diastole**,
- Coexistent CAD

Q. Application of Venturi effect in cardiology – (Gaffar sir)

Venturi effect: Sucking effect of turbulent flow

- Ischaemic chest pain in severe AR
- SAM in HOCM

- LVOTO after Acute anterior MI (due to compensatory hyperkinesis of the basal & mid segments of the LV) – This is uncommon complication of acute anterior MI, which is underappreciated & underreported (Griffin 5e, p.52)

Q. Hypertrophic cardiomyopathy (HCM) – (Griffin 5e, p.130)

- Inspection: JVP – prominent 'a' wave may be present, Precordial heave
- Jerky pulse (bifid carotid pulse – rapid carotid upstroke, followed by a second peak)
- Apex – Laterally displaced, diffuse ; Double apical impulse (S4)
- S1 – normal, preceded by S4
- S2 – normal, or paradoxically split due to prolonged ejection time in severe outflow tract obstruction
- Murmur in neo-aortic area (**tricuspid area**); (as LVOTO, not at the level of AV)
Harsh crescendo-decrescendo systolic murmur at left sternal edge
- Murmur of MR (if HOCM) (If clinically MR, **Obstructive**) (Gaffar sir)
- Associated AR (soft, early decrescendo diastolic murmur) in 10% (Griffin)
- 1/3 HCM: Non-obstructive;
1/3 HCM: Obstructive at rest;
1/3 HCM: Obstructive with provocation (Valsalva); (Aziz bhai) (50% - Griffin)
¼ HCM: Resting gradient between body & LVOT (Griffin)

Q. How to differentiate HCM from HOCM – (Gaffar sir)

- Patient symptomatic
- Murmur of MR

Q. HCM, concentric LVH – What happened to Capillary to myocardial fibre ratio?

Decreased. Muscle hypertrophy, not hyperplasia.

Q. Dynamic auscultation – (1 respiratory cycle ≈ 4 cardiac cycles)

Principles of dynamic auscultation – by altering

- Preload
- Afterload
- Force of contraction (Contractility) (Isoprenaline)
- Heart rate
- ↑Preload → ↑VR → ↑Ventricular size → ↓HCM
- ↓Preload → ↓VR → ↓Ventricular size → ↑HCM.....(Valsalva & standing)
- Squatting → ↑PR & VR → ↑Ventricular size → ↓HCM
- Squatting → ↑PR & VR → ↑SV → ↑AS

Valsalva: force expiration against closed glottis, causes ↑intrathoracic pressure, ↓VR & finally ↓preload

Post-Valsalva release: just reverse of Valsalva, ↑VR

Nitrate: ↓afterload

↑Heart rate: ↓Stroke volume, ↓VR

Conditions that requires dynamic auscultation –

- AS and HOCM
- MVP

Q. Clinically differentialte between AS & HOCM –

	AS (Fixed LVOTO)	HOCM (Dynamic LVOTO)
Site of murmur	Aortic area (ESM)	Neo-aortic area (MSM best heard at the apex & lower left sterna edge.)- Raghwa Rao
Radiation	Towards neck	No radiation
Associated AR	May be present	Never Present (10% - Griffin) (Gaffar sir)
Apex beat	Heaving, sustained	Double apical impulse (Palpable S4 due to LAH)
A2	Soft	Normal
S2		May be single or reversed split
Pulse	Low-volume, slow-rising (Pulsus parvus et tardus) (Parvus=weak, tardus=late)	Jerky (Carotid)
Ejection click	May be present	Absent
Dynamic auscultation of systolic murmur		
Respiration	No change	May increase with expiration
Standing up (↓VR)	Decreases	Increases
Leg raising	Increases	
Valsalva (↓VR, ↓Preload)	Decreases	Increases (Reduction LV volume & LV size)
Inotropes and Vasodilators (nitrate)		Increases
Squatting (↑arterial pressure, VR & SV)	Increases (also S3, S4, right-sided murmur, MS)	Decreases (↑LV size & ↓LVOTO)
Handgrip (Isometric exercise)	Increases	Decreases (↑LV volume)
Cardiac cath		Brockenbrough-Braunwald-Morrow sign in HCM

TABLE 10.2**Effects of Maneuvers or Pharmacologic Intervention to Differentiate Murmur of Hypertrophic Cardiomyopathy from Aortic Stenosis**

Maneuver	Physiologic effect	HCM	AS	MR
Valsalva and standing	Decreases VR, SVR, and CO	↑	↓	↓
Squat and handgrip	Increases VR, SVR, and CO	↓	↑	↑
Amyl nitrite	Increases VR Decreases SVR and LV volume	↑	↑	↓
Phenylephrine	Increases SVR and VR	↓	↑	↑
Extrasystole	Decreased LV volume	↑	↓	No change
Post-Valsalva release	Increased LV volume	↓	↑	No change

AS, aortic stenosis; CO, cardiac output; HCM, hypertrophic cardiomyopathy; LV, left ventricular; MR, mitral regurgitation; SVR, systemic vascular resistance; VR, venous return; ↓, decrease; ↑, increase.

VASTE ↑ HCM (Valsalva, Amyl nitrite, STanding, Extrasystole)

Q. Athlete's heart vs HCM (Differentiation) (Griffin 4e, p.172) (Griffin 5e, p.139, 595)

	HCM	Athlete's heart
Pattern of hypertrophy	Unusual pattern of hypertrophy	Eccentric hypertrophy
LVEDD	<45 mm	>45 mm
Septal thickening	>15 mm	<15 mm
LA enlargement	Yes	No, LA size <40 mm (p.139) (>40mm) (p.595)
Diastolic dysfunction	Yes	No
Abnormal LV filling	Yes	No
Response to deconditioning		Yes, decrease in LV thickness

- **Athlete's heart:** Structural, functional & electrical adaptations that occur with prolonged & intense exercise training. (Griffin 5e, p.595)
- **Differentiating HCM from hypertrophy of athletes:**
Diagnostic uncertainty is greatest when –
 - When maximal diastolic LV wall thickness exceeds the upper limit of normal (12mm) but is less than the defined lower limit of expected hypertrophy (15mm) for HCM
 - Absence of SAM and LVOT obstruction.**If differentiation not possible –**
 - Stop training
 - After several months, ventricular hypertrophy will typically regress in athletes but will persist in HCM.

Q. Normal ECG changes in athletes –

- 1) **Sinus bradycardia** (as low as 30/min, but >30/min) (<30/min is abnormal)
- 2) **Sinus arrhythmia**
- 3) Ectopic atrial & junctional rhythm (i.e. low atrial rhythm)
- 4) **First-degree AV block** (PR interval >200ms, but <400msec)
- 5) **Mobitz type I (Wenckebach) second-degree AV block**
- 6) **Incomplete RBBB** – upto 1/3 of athletes (attributed to RV remodeling & dilatation)
- 7) **Isolated QRS voltage criteria for LVH** (without non-voltage criteria, i.e. LA enlargement, LAD, ST-segment depression, T inversion or pathologic Q waves)
- 8) Early repolarization pattern (ERP) – in up to 90% athletes
- 9) Convex ('domed') ST-segment elevation, followed by T-wave inversion (TWI) in leads V₁₋₄ in black/ African athletes

Borderline ECG changes in athletes – LAE, RAE, RAD, LAD, complete RBBB

Abnormal ECG changes in athletes –

- T inversion
- ST depression
- Pathological Q waves
- Complete LBBB
- QRS >140 ms
- Epsilon wave, Ventricular pre-excitation
- Prolonged QT,
- Brugada Type I
- Profound sinus tachycardia <30/min
- PR ≥400 ms
- Mobitz type II heart block, Complete heart block
- ≥2 PVCs
- Atrial tachyarrhythmias, Ventricular arrhythmias

Q.

- **Permanent cell** – non-dividing – eg, Cardiac muscle, Neuron, Skeletal muscle
- **Stable cell** – limited regeneration – eg. Parenchymal cells – Liver
- **Labile cell** – hair follicle, lining epithelium

Q. Echo of HCM/ Echo of HOCM – PSAX view (Gaffar sir)

- Apical systolic obliteration of LV cavity
- Pin-hole shaped LV cavity
- **2D** – (Luthra, p.88) (Griffin 5e, p.133)
 - Hypertrophy –
 - Asymmetrical septal hypertrophy (ASH) – 60% (IVSd >15mm)
 - Symmetrical ventricular hypertrophy – 30%
 - Apical hypertrophy – 10%
 - **Thickening of IVS** is confined to the base and impinges on LVOT. (Basal anteroseptum) (Aziz bhai)

- o **IVS: LVPW ratio exceeds 1.5** (1.3, Washington manual, p.105) (Wadud sir)
- o Wall thickness > 15mm (13-15 mm is considered borderline)
- o Thickened IVS shows bright echogenicity with speckling in the myocardium due to myocardial fiber disarray
- o **Septal immobility** (Reduced IVS motion with vigorous excursion of LVPW.)
- o LV cavity appears small
- **M-Mode –** (Luthra, p.90)
 - o **Systolic anterior motion (SAM) of AML.** During **mid-systole**, the AML moves anteriorly to coapt with the IVS, This occurs due to **Venturi effect** caused by high velocity in the LVOT. (Wadud sir)
 - o SAM occurs during **later stage of systole** when LV cavity size becomes smaller. SAM causes dynamic LVOT obstruction. Therefore, HOCM is more appropriate.
 - o LVOTO may present at rest or become more pronounced following provocation (prolonged standing, isovolumic exercise (hand-grip), Valsalva manoeuvre or sublingual nitrate). All these methods reduce LV size and thus increase the degree of LVOT obstruction.
 - o Dynamic LVOTO during later part of systole causes mid-systolic AV closure and late-systolic fluttering of the aortic cusps
 - o **Premature closure of AV, reduced rate of closure of MV during mid-diastole, MVP with MR** (Griffin)
- **Doppler echo –**
 - o CW – Increased peak flow velocity across the LVOT ('Broad-blade dagger' shape, late peaking Doppler envelope)
 - o PW with the sample volume in the LVOT – increased velocity to be proximal to the AV.
 - o The high velocity jet – characteristic concave appearance with the peak velocity (V_{max}) coinciding with peak SAM in midsystole. Normally, it peaks in early systole.
 - o LV diastolic dysfunction (Abnormal transmitral inflow spectral trace – A wave is taller than E wave) ($E < A$)
 - o **MR (eccentric, posteriorly directed)**
 - o **Resting gradient >30 mmHg (HOCM)**
 - o **Provocable gradient >50 mmHg** (Griffin)

Q. Prognostic importance of SAM – (Wadud sir)

- Syncope
- HF (MR)
- SCD

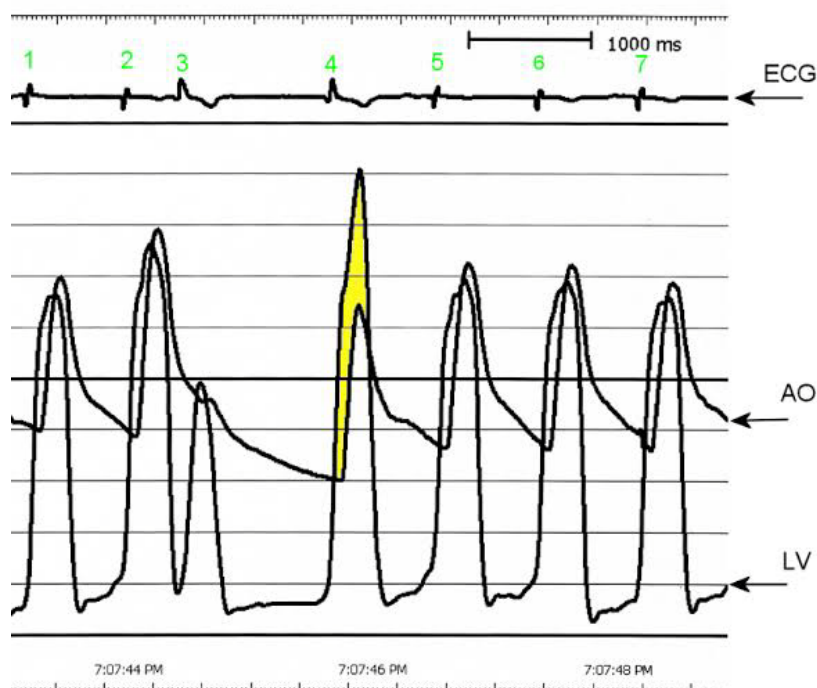
Q. Role of cardiac cath in HCM –

- Define coronary anatomy before procedures for septal reduction or MR, or for the evaluation of ischaemic symptoms
- LV graphy: hypertrophied LV, prominent septal bulge, nearly complete obliteration of the ventricular cavity during systole, SAM, MR
 - o **Spadelike appearance of the ventricular cavity (Apical HCM)**

Q. Brockenbrough-Braunwald-Morrow sign - (Gaffar sir)

- **Severe increase in the LV-aortic gradient in the beat after a PVC because of an increase in contractility & decrease in afterload during post-PVC beat** (Griffin)
- This sign is observed in individuals with HCM with outflow gradient.

- This sign can be used to differentiate HCM from AS.
- In individuals with AS, after a PVC, the following ventricular contraction will be more forceful, and the pressure generated in the LV will be higher. Because of the fixed obstruction that the stenotic AV represents, the post-PVC ascending aortic pressure will increase as well.
- In individuals with HCM, however, the degree of obstruction will increase more than the force of contraction will increase in the post-PVC beat. The result of this is that the LV pressure increases and the ascending aortic pressure **decreases**, with an increase in the LVOT gradient.
- While the Brockenbrough-Braunwald-Morrow sign is most dramatically demonstrated using **simultaneous intra-cardiac and intra-aortic catheters**, it can be seen on routine physical examination as a decrease in the pulse pressure in the post-PVC beat in individuals with HCM.



Q. Management of hypertrophic cardiomyopathy (HCM) –

- **General measures –**
 - Avoid heavy exercise/ physical exertion
 - Avoid dehydration (excessive sunlight exposure)
 - Avoid nitrates, diuretics
(Nitrate ↓preload, ↓afterload → syncope)
- **Medical management –**
 - β -blocker (Not with vasodilating properties like carvedilol, labetalol)
 - Non-DHP CCB (Verapamil, diltiazem)
 - Disopyramide (Class I_a anti-arrhythmic, vagolytic) – typically used in a very symptomatic patients when surgical myectomy/ alcohol septal ablation is planned
 - DHP CCB, ACEi/ ARB – contraindicated.
- **Non-pharmacological Rx/ Intervention/ Surgical management –**
 - Septal myectomy
 - Intervention:
 - Alcohol septal ablation (ASA), risk of conduction abnormalities
 - Dual-chamber PPM (DDD), when severely symptomatic despite optimal medical therapy – (with short AV delay) – to allow RV & IVS to contract early, LV and posterior wall to contract later (Asymmetric contraction)

- Activation of RV apex alters the systolic contraction sequence of basal septum and thus results in reduction in LVOT obstruction.
- RV lead attached to IVS
- ICD
- LV assist device in advanced stage (Khaleq sir)
- Cardiac transplantation (Khaleq sir)

Q. Indications of ICD in hypertrophic cardiomyopathy (HCM) – (Wadud sir)

1. Cardiac arrest survivor or H/O sustained VT
2. Recurrent syncope
3. Family H/O SCD (≥ 1 first-degree relative < 40 years old or any age if relative with an established diagnosis of HCM)
4. AECG – NSVT (≥ 3 consecutive beats at $\geq 120/\text{min}$ for < 30 seconds)
5. Echo – Maximum left ventricular wall thickness - Greatest risk of SCD in patients with a maximum wall thickness of ≥ 30 mm
(Marked increase in LV wall thickness)-Davidson
6. Echo – Dilated LA
7. ETT – Failure to increase systolic pressure by at least 20 mm Hg from rest to peak exercise or a fall of > 20 mm Hg from peak pressure
(Exercise-induced hypotension)- (Davidson)

Q. Causes of dynamic outflow tract obstruction – (Gaffar sir)

- HOCM
- Acute anterior MI (Compensatory hyperkinesis of the basal & mid segments of the LV)
- Takotsubo cardiomyopathy/ Apical ballooning syndrome/ Stress cardiomyopathy
- TOF
- Subvalvular/ infundibular PS

Q. Mitral valve prolapse (MVP) –

- Prolapse exists when either or both of the mitral leaflets protrude $> 2\text{mm}$ beyond the annulus into the LA during systole
- mitral leaflets (tip/body), with or without MR (Aziz bhai)
- Affects 2% population, M=F
- **Primary prolapse:**
 - Results from myxomatous proliferation of the leaflets (abnormal accumulation of mucopolysaccharides)
 - Impaired tensile strength in the chordae, chordal elongation
 - Thickening of the leaflets $> 5\text{mm}$ (**Classic MVP**)
 - **Barlow disease:** in younger, greater annular dilation, more marked leaflet redundancy & prolapse
 - **Fibroelastic deficiency:** in older patients, confined to P_2 , thinning & rupture of chorda
 - MVP is commonly identified in Marfans, Ehlers-Danlos, osteogenesis imperfect
 - Most complications of prolapse, particularly **severe MR**, are associated with **primary prolapse**.
- **Secondary prolapse:**
 - Relatively normal valvular structure
 - Disproportion between leaflet size & LV cavity size
 - Particularly affects younger women, ASD, hyperthyroidism, emphysema, HCM

- Little clinical significance & not usually associated with significant MR
- Mostly asymptomatic. If prolapse causes MR, symptoms of MR; atrial & ventricular arrhythmias;
- Increased incidence of pectus excavatum, straight back, scoliosis
- **Midsystolic click** (at least 0.14s after S1), **mid-late systolic crescendo murmur**
- **Dynamic auscultation:**
 - Conditions that decreases LV size ($\downarrow$ VR, $\uparrow$ contractility, or $\downarrow$ systemic volume):
Earlier prolapse, earlier click, $\uparrow$ duration of murmur
i.e Standing, Valsalva maneuver, dehydration, exposure to amyl nitrate
 - Conditions that increases LV size (squatting, phenylephrine):
Move the click & murmur later in systole.
- Echo:
 - **2D:** >2mm displacement of one or both mitral leaflets into the LA during systole in PLAX or apical long-axis views (caution in A4CH view)
 - **M-mode:** Late or holosystolic bowing of the MV leaflet 3mm or more below the C-D line.

Q. Takotsubo cardiomyopathy/ Apical ballooning syndrome/ Stress cardiomyopathy/ Broken heart syndrome – (Griffin 5e, p.93, 573, 844)

- **Transient LV dysfunction** in the setting of **intense physical or emotional stress**
- Apical ballooning with preserved basal LV systolic function (Japanese Octopus trap)
- **Pathophysiology:** (Catecholamine toxicity) Catecholamine-induced myocardial dysfunction & coronary microvascular dysfunction (CMD); $\pm$ coronary vasospasm (highest proportion of β -receptor at LV apex)
- Most prevalent among post-menopausal women (90% female and >50yr)
- **Often mimic acute MI**; chest pain, **abnormal ECG + positive biomarkers** (mild elevation)
- **ECG:** **Dynamic ST-segment elevation** (anterior ST elevation), and **T inversion**
- **Echo:** Characteristic finding – severe apical hypokinesis or akinesis with relatively preserved function of LV base. **(In most, LV systolic function recovers within 4-6 weeks)**
- **Cardiac MRI:** No perfusion abnormalities & no evidence of delayed gadolinium enhancement. (Useful for differentiating from myocarditis & ACS)
- **CAG:** No obstructive CAD.
- **Rx:** **In acute phase**, Rx is targeted at electrical & mechanical complications resulting from acute LV dysfunction. **In subacute to chronic phase**, medical therapy for LV dysfunction (ACEi/ARB & β -blockers)
- **Prognosis:** Very good. Majority recovers full LV function within weeks to months.
- No proven therapy to prevent recurrence.
- Annual rate of recurrence 2.5-2.9%/yr, most common in younger than 50 yrs
- In-hospital mortality: 2-7% (similar to STEMI)

Q. Causes of lung creps –

- LV systolic dysfunction – MI, myocarditis
- LV diastolic dysfunction
- LA failure – severe MS
- Hyperdynamic circulation
- Volume overload – Renal failure, liver failure, iv fluid
- Lung causes

Q. Short-acting ACEi –

- Enalapril 5, 10 mg – (Anapril, Enaril, Minipril, Eril)
- Captopril 25 mg – (Acetor, Cardopril, Topril)

Q. ACEi (ACE inhibitors) – (Gaffar sir)

- Post-MI
- HF
- LV dysfunction
- Hypertension
- Diabetic nephropathy

Q. RBBB + dialted LA: possibilities

- MS + PH
- ASD + MVP (causing MR)
- MS + ASD (Lutembacher's syndrome)

Q. Chest pain with shock but clear/dry lung - possibilities

- Acute STEMI inferior + RVI
- Acute massive pulmonary embolism
- Acutely developed cardiac tamponade
- Aortic dissection
- Tension pneumothorax

Q. Infective endocarditis – Major criteria – Blood culture positive – meaning? (Atahar sir) (ESC 2015 IE)

1) Typical microorganisms consistent with IE from 2 separate blood cultures:

- Viridans streptococci,
- Streptococcus bovis (Streptococcus gallolyticus)
- HACEK group,
- Staphylococcus aureus; or
- Community-acquired enterococci, in the absence of a primary focus; or

2) Microorganisms consistent with IE from persistently positive blood cultures:

- ≥ 2 positive blood cultures of blood samples drawn >12 h apart; or
- All of 3 or a majority of ≥ 4 separate cultures of blood (with and last samples drawn ≥ 1 h apart); or

3) Single positive blood culture for *Coxiella burnetii* or phase I IgG antibody titre $>1:800$

Q. Culture media for infective endocarditis – (Nazrul sir)

- Glucose broth
- Bile broth
- MacConkey agar (MA)

Q. Drug doses of infective endocarditis (IE) –

- Penicillin G – 12-18 million units/day in 4-6 doses or continuously
- **Ceftriaxone –**
 - o **2 g/day in 1 dose** (Paedi – 100 mg/Kg/day) for Streptococcus

- o **4 g/day i.v. or i.m. in 2 doses**, for enterococcus
- Amoxicillin 100-200 mg/Kg/day iv in 4-6 doses
- **Gentamicin – 3mg/Kg/day, 1 dose iv/im** (single dose to ↓renal toxicity)
- **Vancomycin** in MRSA – **30-60 mg/Kg/day** in 2-3 doses (Wadud sir)
- Ampicillin, Flucloxacillin – 12 g/day iv in 4-6 doses
- **Rifampicin** – 900-1200 mg iv or orally in 2-3 doses (Tab. Firifam 450mg 1+0+1)
(start 3-5 days later than Vancomycin & gentamicin)
- **Streptococcus –**
 - o Pen G/ Amoxi/ Ceftriaxone (4 weeks)
 - o + Genta (if 2 weeks)
 - o Vanco (allergic)
- **Staph** – Flucloxacillin or (Cotrim-Clindamycin)
- **MRSA** in NVE – Vancomycin / Daptomycin / (Cotrim + Clindamycin)
- **MRSA in PVE** – (Flucloxacillin/Vancomycin + Rifampicin + Gentamicin) **VGR**
- **Enterococcus** – (Amoxi/ Ampi + Genta) or Vanco-Genta
- **Empirical Rx for CA-NVE or Late PVE:**
 - o Ampi-Genta-Flucloxacillin or
 - o **Vancomycin + Gentamicin** (for penicillin-allergic patients) **VG** (Wadud sir)
- **Empirical Rx for early PVE: Vancomycin + Gentamicin + Rifampicin VGR**

Antifungal therapy (In ward, Inj. Fluconazole/ Flugal IV 200mg + 100ml NS, iv daily)

- for Candida IE – **Liposomal amphotericin B** (or other lipid formulations) ± flucytosine or an echinocandin at high doses;
- for Aspergillus IE – **Voriconazole** ± (echinocandin/amphotericin B)

Q. Early PVE & late PVE (Prosthetic valve endocarditis) – (Griffin 5e, p.281)

Early PVE –

- Occurring within 2 months of surgery
- Commonly associated with intraoperative contamination & nosocomial infection
 - Coagulase-negative Staphylococci (30%) – Staphylococcus epidermidis
 - Staphylococcus aureus (20%)

Late PVE –

- Onset >2 months after surgery
- Pathogens of native valve IE – *Streptococcus* species, *Staph. aureus*, Enterococcus, Coagulase-negative Staphylococci

Q. Cardiac conditions at highest risk of IE for which Prophylaxis should be considered when a high-risk procedure is performed –

- Any prosthetic valve
- Previous episode of IE
- Congenital heart disease (CHD)
 - o Any cyanotic heart disease

- o Any type of CHD repaired with prosthetic material, upto 6 months after the procedure or lifelong if residual shunt or valvular regurgitation remains
- Prophylaxis of IE for dental procedures requiring
 - o manipulation of the gingival or periapical region of the teeth or
 - o perforation of the oral mucosa
- Recommended prophylaxis - **Antibiotics** –
 - o Amoxicillin or Ampicillin – 2 g orally or IV
 - o Clindamycin – 600 mg orally or IV

Q. IE Rx – one antibiotic must be bactericidal

- Vancomycin: **30-60 mg/Kg/day** in 2-3 doses (Wadud sir)
- Gentamicin: **3mg/Kg/day, 1 dose iv/im** (single dose to ↓renal toxicity)
- Flucloxacillin: 12 g/day (4-6 divided doses)
- Ceftriaxone: **2 g/day in 1 dose** (Paedi – 100 mg/Kg/day) for Streptococcus
4 g/day i.v. or i.m. in 2 doses, for enterococcus

Q. IE prone lesions –

- Most common congenital heart diseases predisposing IE – (Griffin 5e, p.281)
 - o BAV
 - o PDA (Pulmonary side of PDA)
 - o VSD
 - o CoA
 - o TOF
- Vegetations classically occur along the
 - o Line of closure of the valve leaflets.
 - o Atrial surface of incompetent AV valves (MR)
 - o Ventricular surface of the incompetent semilunar valves (AR, AS)
 - o Intracardiac device before endothelialization
- There is no evidence that secundum ASDs increase the risk of IE. (Griffin 5e, p.281)
- MR, AR, AS
- Not ASD, MS

Q. Vegetations – where found (Gaffar sir)

- Perimembranous VSD:
 - o around rim,
 - o **septal leaflet of TV**,
 - o RV free wall

(IE most frequently involves the septal leaflet of TV apparatus at the point of jet impact)

- Muscular VSD: lower incidence of IE, as the jet is attenuated prior to reaching the TV.
- RSOV to RV: around rupture rim, RV free wall
- AV: Ventricular surface
- MV: Atrial surface

Q. Causes of dilated coronary sinus –

- PLSVC (Persistent left SVC)
- Any conditions leading to Right-sided volume overload

- ASD

Q. Pulmonary artery (PA) diameter in echo – Short axis

Upto 21 mm – Normal,
21-27 Gray zone,
>27 mm – dilated

Q. Causes of pulmonary artery dilatation/ Dilated PA – (Washington Manual, Luthra)

- **Right-sided volume overload (VSD, ASD, TR)**
- **Pulmonary hypertension**
- Congenital PA stenosis (Post-stenotic)
- TGA
- Idiopathic dilated PA (rarely)

Q. Echo facts –

- Probe – piezoelectric crystal
- 2D & M-mode, Doppler – Colour, Spectral, tissue
- PW – low velocity, Localization Sample volume
- CW – high velocity
- Short axis – PA –PW – to see PR
- AP4CH - MV – PW – to see LV diastolic dysfunction
- TAPSE – A4C tricuspid lateral annulus - M mode - <17mm(15) is highly suggestive of RV dysfunction
- PA – M-mode on PV leaflet – loss of a dipping (PH)
- HOCM – LVOT gradient, mid-cavity gradient

Q. Echo –

- RV internal dimension 7-23 mm
- RV free wall thickness >5 mm – RVH
- IVC dilated if > 20 mm

Q. Echo – Pseudoaneurysm vs Aneurysm

Pseudo-aneurysm – Narrow neck, posterolateral LV wall, expansile (thin wall, expand in systole), wall – pericardium, friable & liable to rupture, thrombus (clotted haemopericardium) fills cavity, (Luthra)

- LV aneurysm – **wide neck**, at or near LV apex, dyskinetic, wall – **myocardium** – more echogenic, **rupture unlikely**, often associated with pedunculated or laminar thrombus; more common **after anterior MI** (Luthra)

Q. LBBB – Echo

Jerky IVS

Decreased systolic thickening (at least 50%)

Q. LVEF determination – by biplane Simpson

Q. Wall motion abnormalities – BDECHO18

	Changes in thickness	Systolic thickening	Wall motion score
Normal	>30%	>1.5	1
Hypokinesia	10-30%	1.2-1.5	2
Akinesia	<10%	<1.2	3
Dyskinesia	(Paradoxical thinning = bulging)	Thinning	4
Aneurysm	Diastolic deformation	Diastolic deformity	5

Q. IVC –

Diameter	Collapse	RA pressure
<21 mm	>50%	3
>21 mm	<50%	15

Q. Collapsibility index & estimated RA pressure (BDECHO18)

Diameter	Collapse	RA pressure
<17 mm	50%	0-5
>17 mm	>50%	6-10
>17 mm	<50%	10-15
>17 mm	No collapse	>15
<12	Complete collapse	dry

Q. No variation of IVC in which conditions – (Wadud sir)

- Constrictive Pericarditis
- Restrictive cardiomyopathy
- Pericardial tamponade
- IVC is slightly dilated in intubated patients. (↑PEEP, ↑dilatation, ↓variation)

Q. How to understand hydrostatic pressure of a patient – (Wadud sir)

- It is venous pressure, not arterial hypertension.
- Any features of CCF (raised JVP, hepatomegaly), raised hydrostatic pressure
- Direct measurement of CVP
- Echo – IVC diameter & degree of collapsibility
 - o <50% collapse: Abnormal
 - o 50-70% collapse: Normal
 - o >70% collapse: dehydration (Give fluid)

Q. Speckle tracking echocardiography (STE) – Clinical applications

Subclinical systolic dysfunction in hypertensive patients in patients with preserved LVEF using RT3DSTE (Real time 3D STE)

- Myocardial ischaemia, MI, Myocardial viability
- HFpEF
- CRT
- Stress cardiomyopathy, RCM, Constrictive Pericarditis
- Detection of rejection & coronary stenosis in heart transplant patients
- Early detection of chemotherapy-induced cardiomyopathy
- Detection of subclinical disease/ early myocardial involvement

- VHD, congenital heart disease (ASD, TOF)

Myocardial strain imaging is a better investigation of systolic dysfunction prior to visual assessment of wall motion abnormalities or a reduction of measured EF.

RT3DSTE predicts early LV subclinical dysfunction among hypertensive patients with preserved LVEF

Q. Echo features of chamber identification – (Aziz Bhai)

LV –

- Spherical, thick wall, less trabeculation,
- No moderator band
- Septal attachment of AV valve is more cranial & MV has no chordal insertion
- No conus

RV –

- Apical displacement of STV (septal leaflet of TV) <5mm from AML
(Septal attachment of AV valve is more apical & septal surface receives chordal insertion from TV septal leaflet) BDECHO18
- Moderator band
- More trabeculation
- Pyramidal or triangular shape
- Infundibulum present

RA –

- IVC drains here (Subcostal view)
- **IAS – thick limb towards RA** (Valve of foramen ovale towards LA)
(Morphological RA septal wall receives tendinous insertion of Eustachian valve & has **limb of fossa ovalis**) BDECHO18
- Appendage – broad, saddle-shaped
(**RAA – short & stout**) BDECHO18
- Well visualized in PLAX & subcostal sagittal view

LA –

- Usually thumb/finger like appendage when not dilated, broad when dilated (**Long fingerlike**)
- **IAS - Left atrial septal surface** has a **flap of valve of fossa ovalis**. In CHD associated with high RA pressure, eg TA, severe PS, foramen ovale remains open, and flap valve can be seen protruding into the LA
- Pulmonary veins drain here (except TAPVC)
- Well visualized in PLAX & subcostal 4CH view

TV –

- Tricuspid, septophilic
- TV always found in morphologic RV
- In A4CH view, TV is always closer to cardiac apex

MV –

- Bicuspid, septophobic
- MV is always in morphologic LV
- It is farther from cardiac apex
- Parasternal & subcostal short axis view – fish mouth appearance

Chamber identification is needed to see A-V & V-A concordance. (TGA, cTGA)

Q. Aortic regurgitation (AR) – Volume overload, Hyperdynamic state,

- Target – peripheral vasodilation, ↑HR (Upper normal limit) to ↓diastolic time
- Nitrate
- If hypertensive – not β-blockers
 - Good LV function – Nifedipine SR, Amlodipine
 - LV dysfunction – ACEi/ ARB
- **Causes –**
 - Valve incompetence
 - Aortic root dilatation –
 - Marfan's syndrome, EDS, OI
 - Ankylosing spondylitis
 - Syphilis
 - Aortic aneurysm/ dissection involving root
 - Post-stenotic dilatation
 - Age-related/ degenerative
 - Systemic hypertension

Q. Causes of aortic root dilatation – (Gaffar sir)

- Aging
- Atherosclerosis
- HTN
- Aortitis, syphilis
- Marfan syndrome
- Aortic aneurysm
- Aortic dissection involving root

Q. Causes of acute AR – Griffin-253

Leaflet abnormalities	Aortic root or ascending aorta abnormality
Acute IE	
Traumatic rupture	Acute aortic dissection
Acute prosthetic valve dysfunction	Perivalvular leak or dehiscence of prosthetic valves
Post aortic balloon valvuloplasty	

Q. Severe AR – associated with peripheral signs

- Early diastolic murmur
- Austin-Flint murmur (soft mid-diastolic) in apex, S1- not loud here (in contrast to MS)
- Systolic murmur due to increased stroke volume (functional AS)

Q. Peripheral signs of aortic regurgitation – (Gaffar sir)

If peripheral signs are present → Severe AR

- Pulses: (Davidson)
 - Large-volume (Pulsus magnus) or 'collapsing' pulse (Water hammer pulse)
 - Low diastolic & increased pulse pressure
 - Bounding peripheral pulses
 - Quincke's sign: Capillary pulsation in nail beds
 - de Musset's sign: Head nodding with pulse
 - Duroziez's sign : Femoral bruit ('pistol shot')

Upper limb:

- Locomotor brachialis: worm like visible pulsation of the brachial artery
- Quincke's sign/ pulse: visible capillary pulsations. Alternate blanching & reddening in the nail bed can be visualized by transmitting light on the patient's finger tips. The capillary pulsation can also be visualized by pressing a glass slide on the patient's lips.

Lower limb:

- **Duroziez sign** (double murmur) – **more specific** of all peripheral signs of AR.
 - to & fro high volume flow with audible jet during systole & diastole
 - **Proximal occlusion** of femoral artery (2 cm above the stethoscope) – **systolic bruit** (a forward murmur due to powerful contraction of LV & increased stroke volume)
 - **Distal occlusion** of femoral artery (2 cm below the stethoscope) – **diastolic bruit** (a backward murmur due to arterial recoil & back flow)
- **Traube's sign** – pistol-shot murmur (Booming of systolic sounds heard over the femoral artery due to sudden distension of arterial wall.
- **Hill's sign** – **Peak systolic pressure gradient between posterior tibial & radial arteries**. It is due to greater velocity of blood in lower limb artery, which arises from the aorta in a straight course. Severity of AR from Hill's sign may be deduced.
 - Angiographic 2+AR = 20-40 mmHg systolic pressure difference
 - Angiographic 3+AR = 41-60 mmHg systolic pressure difference
 - **Angiographic 4+AR = >60 mmHg pressure difference**

Head & neck:

- **Corrigan's sign** – visible (dancing) carotid pulsation
- **Muller's sign** – Pulsation of the uvula (alternate hyperemia & blanching)

Eye:

- **Becker's sign** – Prominent retinal artery pulsation

Abdomen:

- Rosen bach's sign: pulsations of the liver.
- Gerhardts's sign: pulsation of the spleen.

Q. Causes of suprasternal pulsation – (Gaffar sir)

- Severe AR (Corrigan sign)
- Aneurysm of arch of aorta
- CoA (if 10 yrs female)
- Hyperdynamic circulation (pregnancy, ...)

Q. Cause of acute MR – (Nazrul sir)

- Acute MI – (Ischaemic MR due to PM dysfunction/rupture)
 - Antero-lateral papillary muscle (ALPM) (supplied by LAD + LCX) –
 - Less common but complete rupture of ALPM occurs more frequently than complete rupture of PMPM
 - **Postero-medial papillary muscle** (PMPM) (supplied by PDA or LCX, depending on coronary dominance) –
 - 6-12 times more common than ALPM dysfunction (single blood supply)
 - Partial rupture is more common
 - Common in **inferior MI**
- Acute IE
- Traumatic rupture/ surgery (Post-PTMC)
- Acute rheumatic carditis
- ?Valve dehiscence

Mx of acute MR – diuretics, Rx of underlying cause

Murmur radiation:

- Rupture of **A**nterior leaflet of chorda tendineae → **A**xilla & back
- Rupture of posterior leaflet of chorda tendineae → base & carotid arteries

Causes of MR – according to age (Gaffar sir) (Griffin 5e, p.226)

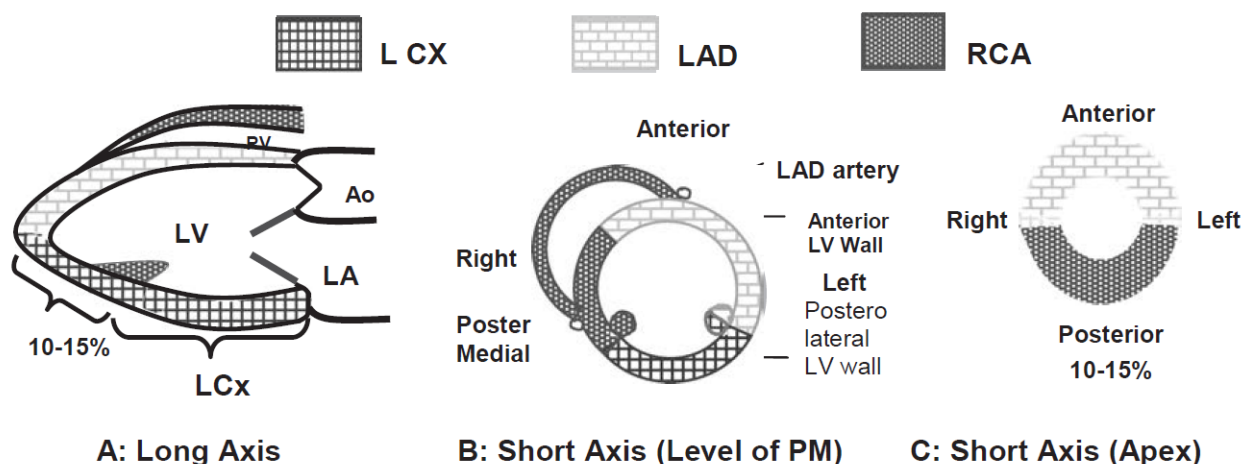
- Neonate – MVP, Cleft mitral leaflet
- Early adolescent – ARF
- Adult – Chronic RHD, IE, Trauma, DCM, IHD
- Elderly –
 - Myxomatous degeneration of leaflets with excessive motion (most common)
 - Mitral annular calcification (MAC)
 - Degenerative disorder, accelerated by HTN & DM
 - Also seen in renal failure with dystrophic calcification, RHD, Marfan syndrome, Hurler syndrome

- **Leaflet abnormalities:** Myxomatous, RHD, IE, Aneurysm, congenital
- **Mitral annular abnormalities**
- **Chordal abnormalities:** Chordal rupture, RHD
- **Papillary muscle abnormalities**

Papillary muscle: (Griffin 5e, p.236)

Anterolateral PM: Blood supply from LAD & LCX.

Posteromedial PM: Blood supply from PDA (RCA/ LCX), depending on coronary dominance.



Q. Description of Mitral stenosis, 2D & M-mode & Doppler echo findings –

MV – Tips & body of both AML & PML thickened, diastolic doming of AML, scattered calcification, restricted opening, both commissures are fused with moderate subvalvular changes.

MVA by planimetry 0.9 cm^2 .

MR – systolic non-coaptation

PLAX view of MS – Luthra p.157

- Diastolic doming of AML
- Thickening of valve leaflets (tips to base → Rheumatic)
(AML, PML – **echogenic, thickened**; don't say calcification) (Wadud sir)
- Restricted opening of the valves
- Dilatation of LA

M-mode scan of MS: Luthra p.159 (2D-guided M-mode)

- Reverberation of echoes from the valve
- Reduced anterior excursion of AML (D-E excursion of the AML is reduced to $< 20 \text{ mm}$ (Normal $20-35 \text{ mm}$))
- Paradoxical anterior motion of the PML
- Flattening of E-F slope of the AML (**Reduced E-F slope**) (Wadud sir)

Doppler echo: (Reduced EF slope) (Wadud sir at exam)

- **PW – A4CH view, sample volume in the LV** – (Luthra)
MV inflow spectral trace shows an **increased peak diastolic flow velocity** exceeding 1 m/s (Normal velocity is $0.6-1.4 \text{ m/s}$; mean 0.9 m/s)
- Slow decay of the velocity with flat slope of deceleration.
Mean pressure gradient across the valve exceeds 4 mmHg . (Luthra)
- CW Doppler of mitral inflow with pressure half-time (PHT) measurement and VTI (Velocity time integral)

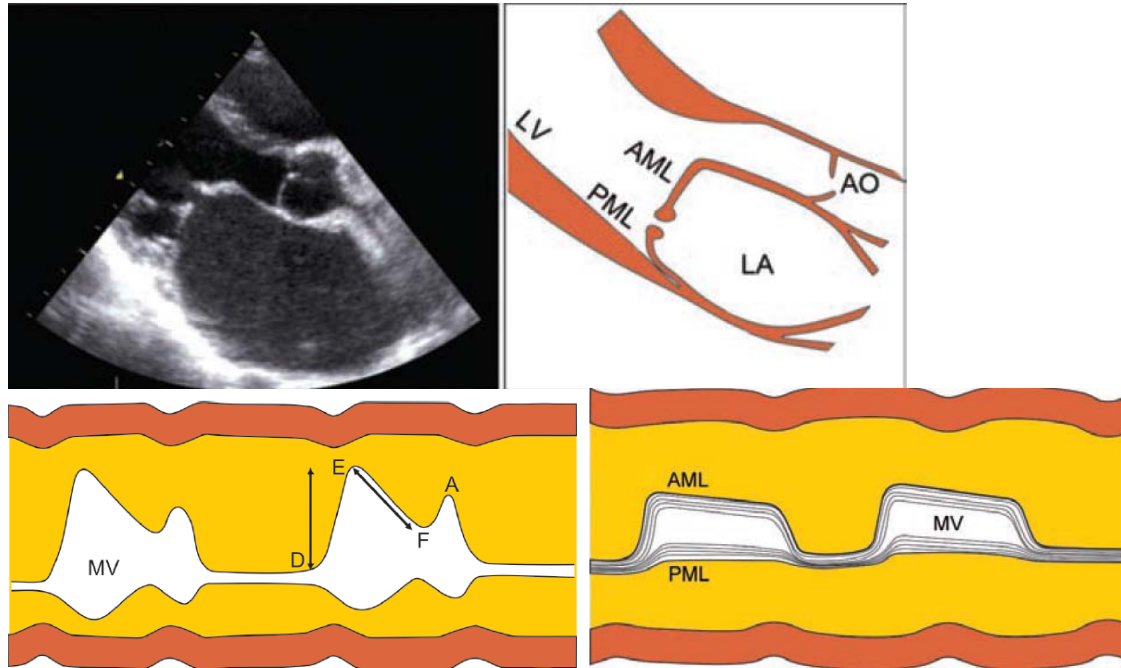
Mean & peak MV gradients: (Washington manual, p.156)

- CW Doppler signal is obtained through the MV in the A4CH view, Doppler profile is traced for calculation of the gradient.
- Peak gradient is calculated using peak velocity in the modified Bernoulli equation,

$$\Delta P = 4 \times v^2$$

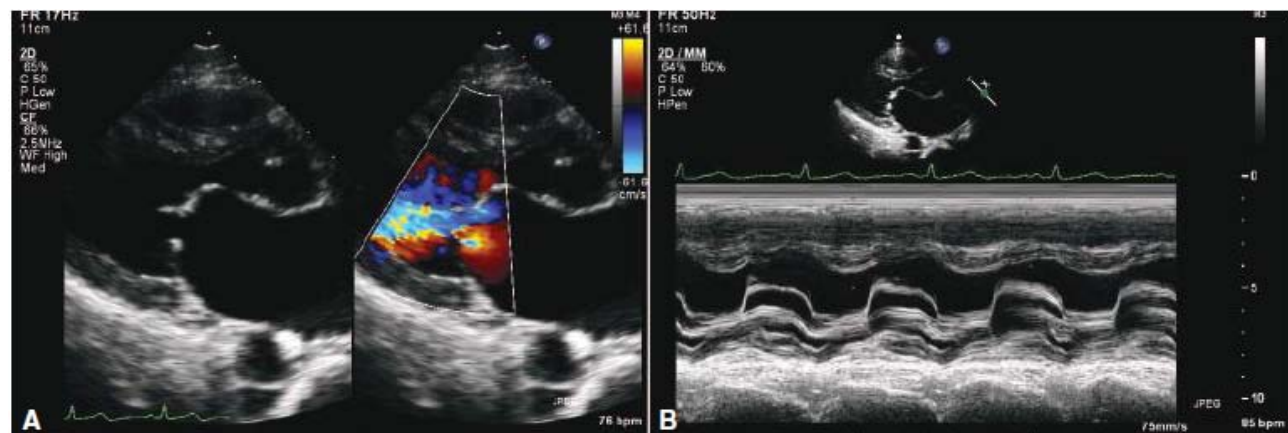
- Mean gradient reflects the average gradient between LA & LV during diastole. It is calculated by averaging the instantaneous gradients (tracing the CW Doppler MV envelope)
- **Mean gradient is more useful clinically;** It is influenced by heart rate, diastolic filling time, CO and associated MR.

PLAX view - 2D

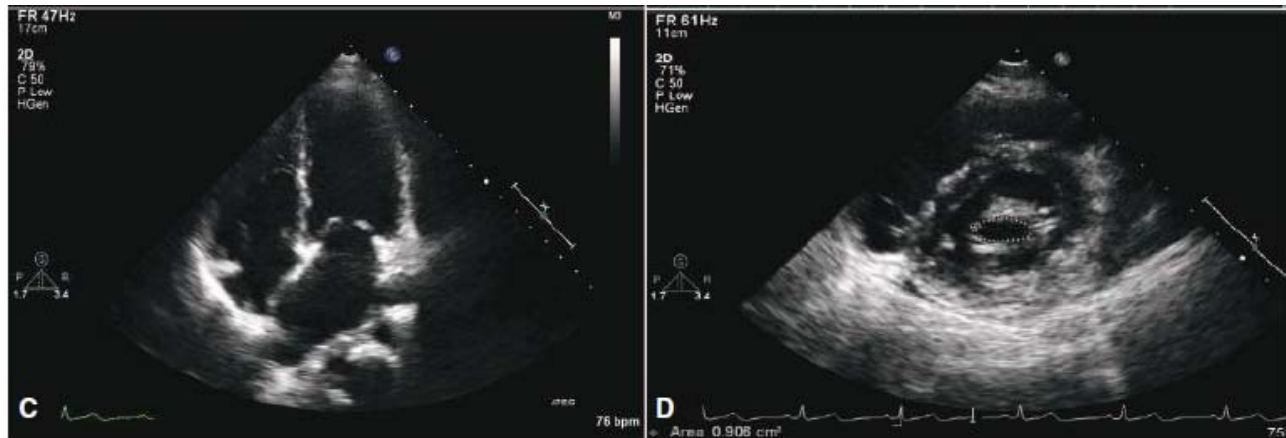


Normal M-mode

Mitral stenosis – M-mode



A. Transthoracic two dimensional echocardiogram parasternal long-axis view demonstrating “hockey-stick” appearance of anterior mitral leaflet. **B.** M-mode echocardiogram across tips of mitral valve leaflets showing reduced E-F slope and paradoxical anterior motion of posterior leaflet.



C. Four-chamber view showing thickening of both leaflets and immobility of posterior mitral leaflet.

D. Planimetry of mitral valve in short axis with valve area of 0.9 cm^2 .

Q. After CMC, Echo – difficult to find LAA appendage (Gaffar sir)

Q. How to see LAA and PA in PSAX – (Khairul bhai)

- LAA: Superolateral
- PA: Inferomedial

Q. Restricted opening of MV due to –

- MS
- $\uparrow$ LVEDP
- AR, regurgitant blood impinge on MV

Q. Normal velocities – (Luthra p.53)

Valve	Range	Mean
Tricuspid inflow velocity	0.3-0.7 m/s	0.50 m/s
Pulmonary outflow velocity	0.5-1.0 m/s	0.75 m/s
Mitral inflow velocity	0.6-1.4 m/s	0.90 m/s
Aortic outflow velocity	0.9-1.8 m/s	1.30 m/s

TV velocity $\times 2$ = MV velocity; PV velocity $\times 2$ = AV velocity

Q. Echo feature of mitral stenosis (MS) severity –

- $\text{MVA} < 1 \text{ cm}^2$
- **Mean PG $> 10 \text{ mmHg}$ (> 12 very severe) Aziz Bhai**
- Pressure half time $> 220 \text{ msec}$
- **Pulmonary pressure $> 30 \text{ mmHg}$**
- Tricuspid flow velocity ($\text{TR } V_{\text{max}}$) $> 3 \text{ m/sec}$

Q. Mitral stenosis severity from pressure half-time (PHT) and transtricuspid velocity – Luthra (MVA normal $4-6 \text{ cm}^2$)

Severity of MS	MVA (cm^2)	P $\frac{1}{2}$ t (msec)	TR V_{max} (m/sec)
Mild	1.5-2.5 (AHA-progressive)	< 150	< 2.7
Moderate	1.0-1.5 (AHA-Severe)	150-220	2.7-3.0
Severe	< 1.0 (AHA-Very severe)	> 220	> 3.0

Q. Stages of mitral stenosis (MS) – AHA/ACC

Stage	Definition	Valve anatomy	Valve haemodynamics
A	At risk of MS	Mild valve doming during diastole	Normal transmitral flow velocity
B	Progressive MS	- Rheumatic valve changes with commissural fusion and diastolic doming of the MV leaflets - Planimetered MVA >1.5 cm²	- Increased transmitral flow velocities - MVA > 1.5 cm² - Diastolic pressure half-time <150 ms
C	Asymptomatic severe MS	- Rheumatic valve changes with commissural fusion and diastolic doming of the MV leaflets - Planimetered MVA ≤1.5 cm² (MVA <1 cm² with very severe MS)	- MVA 1.5 cm² or less (MVA <1 cm² with very severe MS) - Diastolic pressure half-time ≥150 ms - Diastolic pressure half-time ≥220 ms with very severe MS
D	Symptomatic severe MS	- Rheumatic valve changes with commissural fusion and diastolic doming of the MV leaflets - Planimetered MVA ≤1.5 cm²	Same as Asymptomatic severe MS

Table 13. Stages of MS

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of MS	Mild valve doming during diastole	Normal transmitral flow velocity	None	None
B	Progressive MS	- Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets - Planimetered MVA >1.5 cm²	- Increased transmitral flow velocities - MVA >1.5 cm² - Diastolic pressure half-time <150 ms	- Mild-to-moderate LA enlargement - Normal pulmonary pressure at rest	None
C	Asymptomatic severe MS	- Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets - Planimetered MVA ≤1.5 cm² (MVA ≤1.0 cm² with very severe MS)	- MVA ≤1.5 cm² (MVA ≤1.0 cm² with very severe MS) - Diastolic pressure half-time ≥150 ms (Diastolic pressure half-time ≥220 ms with very severe MS)	- Severe LA enlargement - Elevated PASP >30 mm Hg	None
D	Symptomatic severe MS	- Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets - Planimetered MVA ≤1.5 cm²	- MVA ≤1.5 cm² (MVA ≤1.0 cm² with very severe MS) - Diastolic pressure half-time ≥150 ms (Diastolic pressure half-time ≥220 ms with very severe MS)	- Severe LA enlargement - Elevated PASP >30 mm Hg	- Decreased exercise tolerance - Exertional dyspnea

The transmitral mean pressure gradient should be obtained to further determine the hemodynamic effect of the MS and is usually >5 mm Hg to 10 mm Hg in severe MS; however, due to the variability of the mean pressure gradient with heart rate and forward flow, it has not been included in the criteria for severity.

LA indicates left atrial; LV, left ventricular; MS, mitral stenosis; MVA, mitral valve area; and PASP, pulmonary artery systolic pressure.

Q. Echo criteria for severity of Aortic regurgitation (AR) – (Qualitative)

- Colour flow jet width:** Width of the colour jet versus width of the LVOT (<25% = Mild AR; ≥65% = Severe AR)
The length of the AR jet should not be used to assess AR severity.
- Doppler vena contracta width:**
Vena contracta <3mm = mild AR; >6mm = Severe AR
- PHT:** <200 ms = severe AR; >500 ms = mild AR
- Diastolic flow reversal in aorta**

Practically in AR we see – (NHF-2018)

- Jet width at annulus level
- LV dimension
- Holodiastolic flow reversal (at least moderate AR)
- Vena contracta

Q. Echo criteria for severity of Aortic regurgitation (AR) – (Quantitative) Washington manual -

- Proximal isovelocity surface area (PISA)
- Regurgitant volume (R Vol)
- Regurgitant fraction (RF)
- Effective regurgitant orifice area (EROA)

Table 11. Stages of Chronic AR

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AR	• Bicuspid aortic valve (or other congenital valve anomaly) • Aortic valve sclerosis • Diseases of the aortic sinuses or ascending aorta • History of rheumatic fever or known rheumatic heart disease • IE	• AR severity: none or trace	• None	• None
B	Progressive AR	• Mild-to-moderate calcification of a trileaflet valve bicuspid aortic valve (or other congenital valve anomaly) • Dilated aortic sinuses • Rheumatic valve changes • Previous IE	• Mild AR: ◦ Jet width <25% of LVOT; ◦ Vena contracta <0.3 cm; ◦ RVol <30 mL/beat; ◦ RF <30%; ◦ ERO <0.10 cm²; ◦ Angiography grade 1+ • Moderate AR: ◦ Jet width 25%–64% of LVOT; ◦ Vena contracta 0.3–0.6 cm; ◦ RVol 30–59 mL/beat; ◦ RF 30%–49%; ◦ ERO 0.10–0.29 cm²; ◦ Angiography grade 2+	• Normal LV systolic function • Normal LV volume or mild LV dilation	• None
C	Asymptomatic severe AR	• Calcific aortic valve disease • Bicuspid valve (or other congenital abnormality) • Dilated aortic sinuses or ascending aorta • Rheumatic valve changes • IE with abnormal leaflet closure or perforation	• Severe AR: ◦ Jet width ≥65% of LVOT; ◦ Vena contracta >0.6 cm; ◦ Holodiastolic flow reversal in the proximal abdominal aorta ◦ RVol ≥60 mL/beat; ◦ RF ≥50%; ◦ ERO ≥0.3 cm²; ◦ Angiography grade 3+ to 4+; ◦ In addition, diagnosis of chronic severe AR requires evidence of LV dilation	C1: Normal LVEF (≥50%) and mild-to-moderate LV dilation (LVESD ≤50 mm) C2: Abnormal LV systolic function with depressed LVEF (<50%) or severe LV dilatation (LVESD >50 mm or indexed LVESD >25 mm/m²)	• None; exercise testing is reasonable to confirm symptom status
D	Symptomatic severe AR	• Calcific valve disease • Bicuspid valve (or other congenital abnormality) • Dilated aortic sinuses or ascending aorta • Rheumatic valve changes • Previous IE with abnormal leaflet closure or perforation	• Severe AR: ◦ Doppler jet width ≥65% of LVOT; ◦ Vena contracta >0.6 cm, ◦ Holodiastolic flow reversal in the proximal abdominal aorta, ◦ RVol ≥60 mL/beat; ◦ RF ≥50%; ◦ ERO ≥0.3 cm²; ◦ Angiography grade 3+ to 4+; ◦ In addition, diagnosis of chronic severe AR requires evidence of LV dilation	• Symptomatic severe AR may occur with normal systolic function (LVEF ≥50%), mild-to-moderate LV dysfunction (LVEF 40%–50%), or severe LV dysfunction (LVEF <40%); • Moderate-to-severe LV dilation is present.	• Exertional dyspnea or angina or more severe HF symptoms

AR indicates aortic regurgitation; ERO, effective regurgitant orifice; HF, heart failure; IE, infective endocarditis; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; LVOT, left ventricular outflow tract; RF, regurgitant fraction; and RVol, regurgitant volume.

Q. Echo criteria of mitral regurgitation (MR) –

	Mild MR	Moderate MR	Severe MR
Jet area (cm ²)	<4	4-8	>8
% of LA area (Washington/ Luthra)	<20% (<25%)	20-40% (25-50%)	>40% (>50%)
Vena contracta	<3mm		≥7mm

(eccentric MR)			
PW	< 2cm into LA	upto middle LA	upto distal LA

PISA (MR)

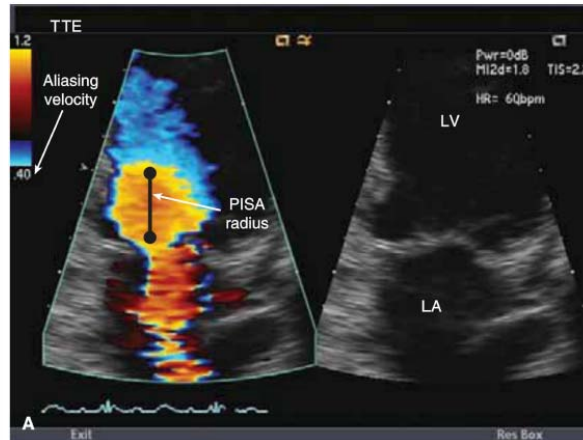
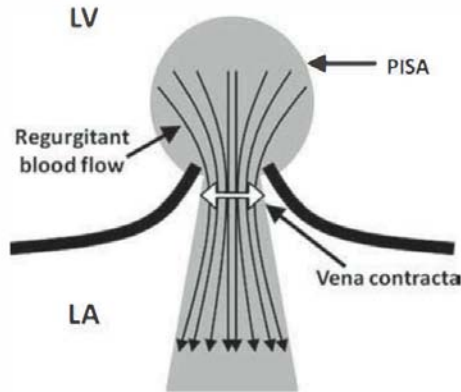


Table 15. Stages of Primary MR

Grade	Definition	Valve Anatomy	Valve Hemodynamics*	Hemodynamic Consequences	Symptoms
A	At risk of MR	- Mild mitral valve prolapse with normal coaptation - Mild valve thickening and leaflet restriction	- No MR jet or small central jet area <20% LA on Doppler - Small vena contracta <0.3 cm	None	None
B	Progressive MR	- Severe mitral valve prolapse with normal coaptation - Rheumatic valve changes with leaflet restriction and loss of central coaptation - Prior IE	- Central jet MR 20%–40% LA or late systolic eccentric jet MR - Vena contracta <0.7 cm - Regurgitant volume <60 mL - Regurgitant fraction <50% - ERO <0.40 cm² - Angiographic grade 1–2+	- Mild LA enlargement - No LV enlargement - Normal pulmonary pressure	None
C	Asymptomatic severe MR	- Severe mitral valve prolapse with loss of coaptation or flail leaflet - Rheumatic valve changes with leaflet restriction and loss of central coaptation - Prior IE - Thickening of leaflets with radiation heart disease	- Central jet MR >40% LA or holosystolic eccentric jet MR - Vena contracta ≥0.7 cm - Regurgitant volume ≥60 mL - Regurgitant fraction ≥50% - ERO ≥0.40 cm² - Angiographic grade 3–4+	- Moderate or severe LA enlargement - LV enlargement - Pulmonary hypertension may be present at rest or with exercise C1: LVEF >60% and LVESD <40 mm C2: LVEF ≤60% and LVESD ≥40 mm	None
D	Symptomatic severe MR	- Severe mitral valve prolapse with loss of coaptation or flail leaflet - Rheumatic valve changes with leaflet restriction and loss of central coaptation - Prior IE - Thickening of leaflets with radiation heart disease	- Central jet MR >40% LA or holosystolic eccentric jet MR - Vena contracta ≥0.7 cm - Regurgitant volume ≥60 mL - Regurgitant fraction ≥50% - ERO ≥0.40 cm² - Angiographic grade 3–4+	- Moderate or severe LA enlargement - LV enlargement - Pulmonary hypertension present	- Decreased exercise tolerance - Exertional dyspnea

*Several valve hemodynamic criteria are provided for assessment of MR severity, but not all criteria for each category will be present in each patient. Categorization of MR severity as mild, moderate, or severe depends on data quality and integration of these parameters in conjunction with other clinical evidence.

ERO indicates effective regurgitant orifice; IE, infective endocarditis; LA, left atrium/atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; and MR, mitral regurgitation.

Q. Echo features of aortic stenosis (AS) severity – (AHA/ACC 2014)

Table 8. Stages of Valvular AS

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AS	- Bicuspid aortic valve (or other congenital valve anomaly) - Aortic valve sclerosis	Aortic $V_{max} < 2$ m/s	None	None
B	Progressive AS	- Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or - Rheumatic valve changes with commissural fusion	- Mild AS: Aortic V_{max} 2.0–2.9 m/s or mean $\Delta P < 20$ mm Hg - Moderate AS: Aortic V_{max} 3.0–3.9 m/s or mean ΔP 20–39 mm Hg	- Early LV diastolic dysfunction may be present - Normal LVEF	None
C: Asymptomatic severe AS					
C1	Asymptomatic severe AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	- Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg - AVA typically is ≤ 1.0 cm² (or AVAi ≤ 0.6 cm²/m²) - Very severe AS is an aortic $V_{max} \geq 5$ m/s or mean $\Delta P \geq 60$ mm Hg	- LV diastolic dysfunction - Mild LV hypertrophy - Normal LVEF	None: Exercise testing is reasonable to confirm symptom status
C2	Asymptomatic severe AS with LV dysfunction	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	- Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg - AVA typically ≤ 1.0 cm² (or AVAi ≤ 0.6 cm²/m²)	LVEF $< 50\%$	None
D: Symptomatic severe AS					
D1	Symptomatic severe high-gradient AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	- Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg - AVA typically ≤ 1.0 cm² (or AVAi ≤ 0.6 cm²/m²) but may be larger with mixed AS/AR	- LV diastolic dysfunction - LV hypertrophy - Pulmonary hypertension may be present	- Exertional dyspnea or decreased exercise tolerance - Exertional angina - Exertional syncope or presyncope
D2	Symptomatic severe low-flow/low-gradient AS with reduced LVEF	Severe leaflet calcification with severely reduced leaflet motion	- AVA ≤ 1.0 cm² with resting aortic $V_{max} < 4$ m/s or mean $\Delta P < 40$ mm Hg - Dobutamine stress echocardiography shows AVA ≤ 1.0 cm² with $V_{max} \geq 4$ m/s at any flow rate	- LV diastolic dysfunction - LV hypertrophy - LVEF $< 50\%$	- HF - Angina - Syncope or presyncope
D3	Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS	Severe leaflet calcification with severely reduced leaflet motion	- AVA ≤ 1.0 cm² with aortic $V_{max} < 4$ m/s or mean $\Delta P < 40$ mm Hg - Indexed AVA ≤ 0.6 cm²/m² and - Stroke volume index < 35 mL/m² - Measured when patient is normotensive (systolic BP < 140 mm Hg)	- Increased LV relative wall thickness - Small LV chamber with low stroke volume - Restrictive diastolic filling - LVEF $\geq 50\%$	- HF - Angina - Syncope or presyncope

AR indicates aortic regurgitation; AS, aortic stenosis; AVA, aortic valve area; AVAi, aortic valve area indexed to body surface area; BP, blood pressure; HF, heart failure; LV, left ventricular; LVEF, left ventricular ejection fraction; ΔP , pressure gradient; and V_{max} , maximum aortic velocity.

	Aortic sclerosis	Mild AS	Moderate AS	Severe AS
Aortic jet velocity (m/sec)	2.5 or less	2.6-2.9	3.0-4.0	>4.0
Mean gradient (mmHg)		<20	20-40	>40
AVA (cm²)		>1.5	1.0-1.5	<1.0
AV opening (ACS) (mm)		13-15	8-12	<8
Indexed AVA (cm²/m²)		>0.85	0.6-0.85	<0.6
Velocity ratio		>0.50	0.25-0.50	<0.25

Normal peak aortic systolic outflow velocity 0.9-1.8 m/s (In AS, >2m/s) (Washington manual)

Q. Defining aortic stenosis (AS) severity (AHA)

Indicator	Mild	Moderate	Severe
Jet velocity	<3 m/s	3-4	≥4 m/s
Mean gradient	<25 mmHg	25-40	>40 mmHG
Peak gradient			
Valve area	>1.5 cm ²	1.0-1.5 cm ²	< 1.0 cm ² (<0.6 cm ² /m ²)

Q. Pulse of aortic stenosis –

- **Pulsus tardus et parvus/ Pulsus parvus et tardus/ slow-rising pulse/ anacrotic pulse**
- The pulse is weak/ small (parvus), and late (tardus) relative to its expected characteristics (**delayed slow-rising carotid upstroke**)
(Aortic regurgitation – Water-hammer pulse)

Q. Prognosis of severe aortic stenosis if no intervention done – (ASD – 532)

- Angina – from the first attack, 5 year survival 50%, without surgical intervention
- Syncope – from the first attack, 3 year survival 50%, without surgical intervention
- Dyspnoea/ Failure – from the first attack, mean survival < 2years, if treated medically

Q. Natural history of AS – how it differs from other VHD – (Atahar sir)

- AS – long latent phase (asymptomatic), near-normal survival
- When symptoms of AS develop, the survival rate decreases markedly, unless AVR
(Once symptomatic, urgent Rx is necessary)

Q. Calculation of AVA based on 'Continuity principle' – (Griffin 5e, p.204)

- Flow of an incompressible fluid in a closed system must remain constant.
- Flow in a vessel is the product of the cross-sectional area (A) of the vessel and the velocity (V).
- Area (A) = $\pi R^2 = \pi D^2/4 = 0.785D^2$, where R=radius of the vessel & D=diameter.
- $A_1V_1 = A_2V_2$; $A_2 = A_1V_1 / V_2$
- $\text{Area}_{\text{aortic valve}} = 0.785 \times \text{diameter}_{\text{LVOT}}^2 \times \text{VTI}_{\text{LVOT}} / \text{VTI}_{\text{aortic valve}}$
- VTI= time-velocity integral
- The continuity equation is valid only for valvular AS
- It cannot be used to assess valve area when there are stenoses in series such as valvular & subvalvular narrowing occurring simultaneously.

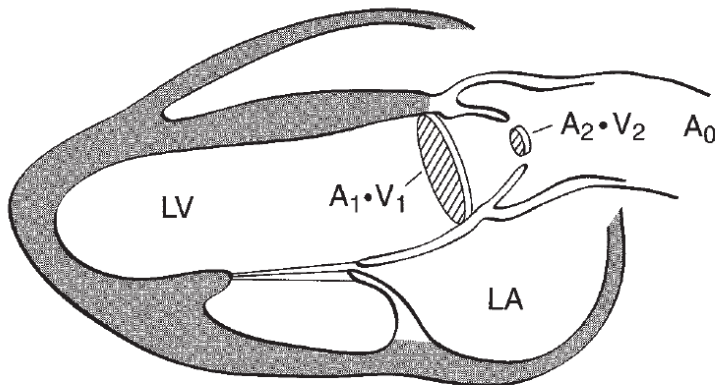


FIGURE 15.4 A schematic representation of parasternal long-axis view and the continuity principle.

Q. Low-flow, low-gradient severe AS – (Griffin 5e, p.213)

- True anatomically severe AS ($AVA < 1 \text{ cm}^2$) and low transvalvular gradient ($MPG < 30 \text{ mmHg}$)
- **DSE:** $\uparrow CO$, $\uparrow$ transvalvular pressure gradient, calculated $AVA \uparrow$ by $< 0.3 \text{ cm}^2$ with $AVA < 1.0 \text{ cm}^2$
- Very poor prognosis without surgery (TAVR – procedure of choice, if feasible)
- Survival depend upon **contractile reserve** ($\uparrow CO$ by 20% from baseline)
- Digoxin is contraindicated in low-flow low gradient AS.

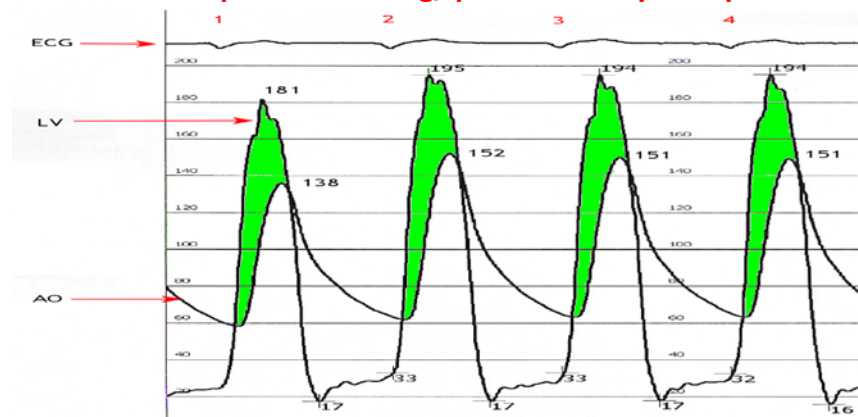
Q. Pseudosevere AS –

- Inappropriately low $AVA < 1.0 \text{ cm}^2$ due to impaired LV function & submaximal opening of mild-to-moderate AS
- **DSE:** $\uparrow CO$, without a significant $\uparrow$ in transvalvular pressure gradient; calculated $AVA \uparrow$ by $\geq 0.3 \text{ cm}^2$ with $AVA > 1.0 \text{ cm}^2$
- Digoxin can be given. (Digoxin is contraindicated in outflow tract obstruction.)

Q. Paradoxical AS –

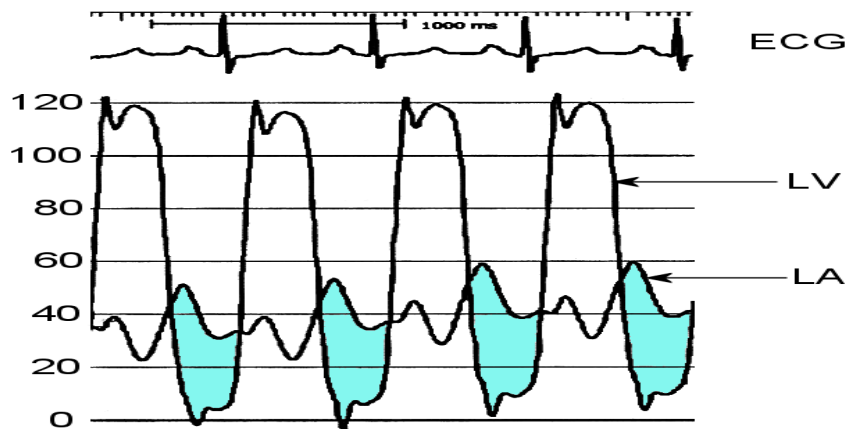
- Paradoxical AS with preserved systolic function with reduced stroke volume index ($< 35 \text{ ml/m}^2$) due to marked LVH and small LV size.

Q. Describe the pressure tracing/ pressure study with possible diagnosis –



Pressure tracing of aortic stenosis.

Q. Describe the pressure tracing/ pressure study with possible diagnosis –



LV pressure – normal, LA pressure – elevated.

Dx: Mitral stenosis

Q. How to write paediatric echo report –

- Description of cardiac lesion – e.g situs solitus
- Measurement of cardiac chambers & vessels (A-V & V-A concordance)
- Evaluation of valves & gradients
- Evaluation of cardiac functions
- Wall motions abnormalities
- Conclusion & recommendations

(Chloral hydrate/ Pedichlor, 50-100mg orally may be used as sedative) (NHF-2018)

(Congenital heart disease – knee flexed)

Q. Echo of pulmonary stenosis – (2014 ACC/AHA) (Griffin 5e, p.258)

Severity	Peak jet velocity (m/s)	Peak gradient (mmHg)
Severe PS	>4	>64
Moderate PS	3-4	36-64
Mild PS	<3	<36

The peak gradient is measured across the PV by using **CW Doppler** with the modified Bernoulli equation.

Q. Patient with severe pulmonary stenosis may present with cyanosis & clubbing –

- Due to associated PFO (leading to right to left shunting) (Gaffar sir)

Q. Rx of Valvular Pulmonary stenosis (PS) –

- Pulmonary valvuloplasty (**Mensfield balloon**, increase pulmonary annulus 25%)
(NIH catheter to access RV for RV graphy to determine the location of PV, to confirm the size of pulmonary annulus as well as morphology of RVOT)
- Pulmonary valve replacement only if due to carcinoid syndrome (Griffin)
- CAG also can be done earlier

Complications of Balloon pulmonary valvuloplasty (BPV):

- Cardiac perforation leading to pericardial effusion ± tamponade
- Conduction system injury
- Thromboembolic events
- Arrhythmias

- PR
- Valvular tearing or trauma
- Restenosis
- Severe hypotension (**Suicidal RV**) due to subvalvular dynamic obstruction (Khaleq sir)
- Complications related to vascular access & dye, infection & inflammation

Q. Indications of intervention in congenital PS – (ACC/AHA Guidelines) (Griffin 5e, p.259)

- **Class I:** Balloon valvuloplasty is indicated in
 - symptomatic patients with a domed valve (trileaflet valve with fused commissure) and peak gradient of >50 mmHg or
 - >60 mmHg in asymptomatic patients
 - **Class II:** Balloon valvuloplasty may be reasonable in patients with dysplastic valve (Noonan syndrome)
 - if the peak instantaneous Doppler gradient is >50 mmHg in symptomatic or
 - >60 mmHg in asymptomatic adolescents or young adults
 - **Class III:** Balloon valvuloplasty is not indicated in symptomatic patients whose peak instantaneous gradient by Doppler is <30 mm Hg or asymptomatic patients with a peak gradient <50 and normal cardiac output.
-
- Mild to moderate PS (PPG ≤50 mmHg) – well tolerated, do not require surgical or percutaneous intervention.
 - Asymptomatic severe PS and PPG ≥60 mmHg or MPG ≥40 mmHg should undergo intervention to reduce the severity of the stenosis.
 - Symptomatic patients with a PPG ≥50 mmHg or MPG ≥30 mmHg should undergo intervention. (Hurst p.1381)
 - Noonan syndrome – dysplastic and stenotic PV

Q. When pulmonary valvuloplasty is successful – (Wadud sir)

- If transvalvular gradient reduce by at least 50%

Q. Suicidal RV/ Suicide ventricle – (Hurst 14e, p.1382)

After transcatheter balloon pulmonary valvuloplasty –

subvalvular infundibular hypertrophy often results in a dynamic subvalvular gradient across the infundibulum that often increases after valvuloplasty because of the relief of downstream obstruction.

β-blocker therapy before and after valvuloplasty helps decrease this dynamic gradient and avoid the rare, but catastrophic, severe infundibular stenosis characterizing the ‘suicide ventricle’.

Q. Severe PS with reverse shunt vs TOF – (Satpathy, p.205)

	<i>Severe PS with reverse shunt</i>	<i>TOF</i>
General appearance	Moon like (pink) facies, arachnodactyly	No specific type
History of squatting and cyanotic spell	Absent	Cyanotic spell and squatting common complaints
Cyanosis	Mild to moderate may appear only on exertion	Cyanosis even at rest
JVP	Giant a-wave	Normal JVP
Parasternal heave	Present	Absent
Systolic thrill	Present	Absent
Second heart sound	Normal with delayed and diminished P ₂	Second sound is single (only A ₂)
S ₄	Audible (due to increased force of RA contraction)	Not audible
Ejection click	Inconstant Pulmonary ejection click	No pulmonary click (aortic EC present)
Murmur	Harsh ejection systolic murmur more than grade 3/6	Ejection systolic murmur less than grade 3/6
Roentgenography	RA and RV enlargement, post stenotic dilatation of PA	Coeur en Sabot, pulmonary bay concave (boot shaped heart)
ECG	RVH with strain pattern (tall R in V ₁), RA enlargement	RVH with early transition. No RA enlargement

In severe PS, increase right atrial pressure may stretch the foramen ovale leading to a right to left shunt resulting in mild, and rarely, severe cyanosis, thereby raising a differential diagnosis of TOF at bedside.

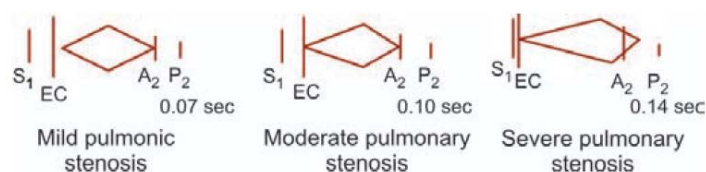


Fig. 22.2: Shape and duration of murmur, S₁-EC, A₂-P₂ interval and intensity of P₂ according to the severity of pulmonary stenosis

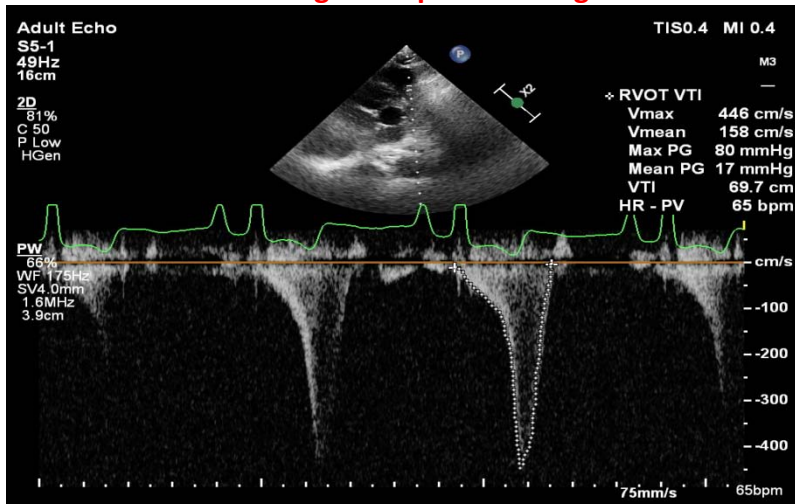
- **Post-stenotic dilatation of the main pulmonary artery (MPA)** is the rule and is usually accompanied by disproportionate dilatation of the **left branch of MPA**. It is due to high velocity (turbulence setup by the jet) of blood ejected through the narrow orifice. Interestingly, the degree of post-stenotic dilatation is **not proportional** to the severity of pulmonary stenosis.
- **Subvalvular pulmonary stenosis** may be infundibular or sub-infundibular.
- **Infundibular pulmonary stenosis** is caused by an anterior and rightward malalignment of the infundibular septum, and occurs in association with a VSD (usually associated with TOF).
- Infundibular hypertrophy in cases of severe PS may produce a second stenosis below the valve. Isolated infundibular stenosis (without VSD) is extremely rare.

Severe PS:

- Pulse is of low volume, JVP shows giant a-wave (right atrial presystolic gallop) and it is raised with onset of RV failure.
- A sustained left parasternal heave is present, **apical impulse is diffuse (RV type)**, systolic thrill over the pulmonary area and presystolic pulsation of liver is often palpable.
- On auscultation **second heart sound is widely split with decreased intensity of P2 (sometime absent) and very often right ventricular S4** is present due to forceful atrial contraction. Right ventricular S3 if present indicates RV failure. If there is no RV failure but S3 it indicates associated ASD or PAPVC.

- In severe PS, **pulmonary ejection click may be absent, or it is very close to S1** because increased RV pressure keeps the pulmonary valve open before ventricular systole. **Ejection systolic murmur is very loud grade 5-6/6 and long with late systolic peak over pulmonary area and also audible over whole of the precordium.**
(Inconstant EC audible over left upper sterna border in mild PS & moderate PS)

Q. Describe the echo findings with possible diagnosis. Name 2 associated conditions.



2D-guided spectral Doppler in PSAX view – Sample volume placed across the RVOT showing - dagger-shaped spectral signal below the baseline. (Aziz Bhai)

Dx: Subvalvular PS.

- 2 Associated conditions:** TOF, VSD

Q. Echo findings in pulmonary embolism – (Washington Manual)

- Free floating thrombus in RV or 'clot in transit' – very rare, but diagnostic of PE (main PA/ saddle embolism)
- PLAX – RVEDD/LVEDD >0.7
- RV enlargement, free wall hypokinesia
 - Abrupt rise of PA pressure
 - McConnell sign:** Normal or Hyperdynamic motion of the RV apex, with akinesia of the RV mid free wall; high specificity for PE
 - Paradoxic IVS:** D-shaped LV with IVS flattening
- 60/60 sign:** RV systolic pressure <60 mmHg + RVOT sceleration time <60s
(CW through TV – peak TR gradient <60 mmHg,
PSAX PW Doppler in the RVOT – acceleration time)

Q. Causes of paradoxic IVS motion – (Luthra p.130)

- LBBB
- Old septal infarction (Old MI anteroseptal)
- Pulmonary hypertension
- Constrictive Pericarditis
- Cardiac surgery

Q. RV size (RV dimensions) & RV function – Echo, qualitative & quantitative

- RV – thin-walled, **pyramidal** structure that 'wrap around' the LV and it appears **triangular** when viewed in cross section
- A4CH view to assess RV size compared with that of the LV – (Qualitative)

- o **Mildly enlarged:** RV is enlarged but $< LV$
 - o **Moderately enlarged:** $RV \approx LV$
 - o **Severely enlarged:** $RV > LV$; apex of the heart comprised of RV
- RV dilation: A4CH view, ensuring the crux & apex, in diastole (Quantitative)
 - o **RV basal diameter $>42\text{mm}$,**
 - o **RV diameter in midlevel $>35\text{mm}$**
 - o **Longitudinal dimension $>86\text{mm}$**
 - o RVOT PLAX proximal diameter $>33\text{mm}$
 - o RVOT PSAX distal diameter $>27\text{mm}$
- RV internal dimension 7-23 mm (Luthra)
- **RV free wall thickness** – measured at peak of R wave on ECG at level of TV chordate in subcostal view (Normal $\leq 5\text{mm}$) (Washington Manual)

M-mode assessment:

TAPSE is the difference in the displacement of the RV base during diastole & systole.

TAPSE (Tricuspid Annular Plane Systolic Excursion) – A4CH view, M-mode cursor is oriented to the junction of the TV plane with the RV free wall.

- o Abnormal excursion is $<16\text{mm}$. (Latest Washington Manual p.59)

TAPSE $<17\text{mm}$ is highly suggestive of RV systolic dysfunction. (ASE guidelines & Older Washington Manual)

- o 12- $<17\text{mm}$ → Mild dysfunction
 - o 8-12 mm → Moderate dysfunction
 - o $<8\text{ mm}$ → Severe dysfunction
 - o **Roughly, $RVEF = TAPSE \times 3$** (Khairul bhai)
- TAPSE – low sensitivity but high specificity

Doppler Assessment: Myocardial performance index (MPI) or Tei index

- The ratio of isovolumic time to ventricular ejection time.
- A measure of both systolic and diastolic function

Q. RA size – upto 44 mm (annulus to annulus) (Mohiuddin)

M-mode, PLAX, AV level

- Aortic root diameter 20-37 mm
- Aortic cusp separation 15-26 mm
- **LA size diameter 19-40 mm**

Q. Normal aortic dimension – (Luthra p.132) (ASE Guidelines)

- Aortic annulus (mid-systole) 17-25 mm (M **26 ± 3** , F **$23 \pm 3\text{mm}$**) ($13 \pm 1\text{ mm/m}^2$)
- Sinus of Valsalva: 22-36 mm (M **34 ± 3** , F **$30 \pm 3\text{mm}$**) (M 17 ± 2 , F $18 \pm 2\text{mm/m}^2$)
- Sinotubular junction: 18-26 mm (M **29 ± 3** , F **$26 \pm 3\text{mm}$**) ($15 \pm 2\text{ mm/m}^2$)
- Aortic root width/ Proximal ascending Ao: 20-37 mm
(M **30 ± 3** , F **$27 \pm 3\text{mm}$**) (M 15 ± 2 , F $16 \pm 3\text{mm/m}^2$)

- **Aortic annulus** - measure at **mid-systole**, inner edge to inner edge
- All other aortic root measurements – measure at **end-diastole**
- Accurate measurement of aortic annulus before TAVI or TAVR is crucial.

Q. LVOT measurement –

- PLAX view – zoom mode
- Measure 5 -10 mm from aortic annulus
- **Mid-systole** (also aortic annulus)
- <18 mm: Narrow LVOT

Q. Spectral Doppler echo – Aziz bhai

- Severe regurgitation/ mild stenosis – triangular
- Severe stenosis/ mild regurgitation – parabolic (half)

Q. Ventricular remodeling –

A disproportionate **thinning**, scarring & **dilatation of the ventricles**. (Aziz bhai)

- Aneurysmal remodeling (figure of 8)
- Spherical remodeling

Infarct expansion – A disproportionate thinning & dilatation of the infarct zone.

Q. β -blockers use to increase BP – (Gaffar sir)

- Any tachyarrhythmias, provided LV function is normal
- Mitral stenosis (also prevent atrial tachyarrhythmia)

Q. Tetralogy of Fallot (TOF) presentation – Growth retardation,

Signs –

- Cyanosis,
- Clubbing,
- **Suffused conjunctiva** (Nazrul sir)

Characteristic finding on palpation/ auscultation – Silent precordium (Gaffar sir)

In contrast, Eisenmenger syndrome has **Hyperdynamic precordium**

Q. Role of brachial BP measurement in TOF – (Satpathy, p.262)

- In TOF, there is triangle of equilibrium (Ao, LV & RV) (Gaffar sir)
- It gives accurate estimate of RVOT gradient
- **RVOT gradient = SBP (brachial) – PASP (15-25 mmHg)**
- Brachial artery systolic pressure = LV systolic pressure = RV systolic pressure (due to large VSD)
- In TOF, PA pressure is invariably normal (15-25 mmHg). Therefore, deducting this value from brachial systolic pressure, gives an approximate of RVOT gradient.

Q. Which component of TOF determines the clinical presentation –

Severity of RVOT obstruction (Pulmonary stenosis)

- Infundibular obstructions (50%)
- Valvular obstruction (20-25%)
- Infundibular & valvular obstruction (20-25%)
- Supravalvular and pulmonary arterial stenosis (rare)

Q. VSD of TOF – (Gaffar sir) (Mir Jamal sir) (Wadud sir)

- Large, peri-membranous, **malaligned**, non-restrictive VSD

- Malalignment due to overriding of aorta (<50%)
- Aorto-septal discontinuity but aorto-mitral continuity is maintained

Q. Echo of TOF – (NHF-2018) (Mir Jamal sir)

- PLAX – echo-dropout in membranous part of IVS, RVH (Wadud sir)
- **Overriding of aorta <50%** (DORV overriding >50%) (?Normally 30% overrides)
- **Aorto-mitral continuity** (DORV – Aorto-mitral discontinuity, except TGA type)
- Conal septum deviation – deviated to laterally & superiorly, causing obstruction (Conal tissue – seen in PSAX view)

Q. Conditions leading to cyanotic spell in TOF –

- Dehydration (diarrhoea, vomiting, **diuretics**)
- Infection (H/O Fever)
- Physical exertion

Q. Management of Cyanotic spell in TOF –

- High-flow oxygen
- Knee-chest positioning
- **NO DIURETICS OR VASODILATORS**
- IV fluid/ Normal Saline
- IV antibiotics
- IV morphine (Pulmonary vasodilation)
- IV sodibicarb (50 ml tds)
- IV β -blocker (IV Propranolol), (if not available, paste the tablet) (prevent dynamic obstruction)
- Emergency pulmonary valvuloplasty (if Valvular PS)

Q. Role of β -blocker in TOF & any other congenital heart disease other than Eisenmenger syndrome –

To improve pulmonary flow, to prevent infundibular spasm
(No β -blocker in DORV, as there is no PS, no infundibular spasm)

Q. Management of TOF –

- General Mx –
- Medical palliative Rx
 - β -blocker,
 - Anti-platelet (Clopidogrel),
NOT ASPIRIN (may precipitate gout due to $\uparrow$ Uric Acid)
 - Isovolemic venesection (300-300-300 ml)
- Surgical palliative Rx –
 - Modified BT shunt (Subclavian artery & ipsilateral pulmonary artery)
 - Waterstone & Potts - obsolete
- Total corrective surgery –
 - Pericardial patch closure of VSD
 - Resection of infundibular tissue (Pulmonary valvuloplasty)

Q. Before isovolemic venesection, what investigation should be done – (Gaffar sir)

- PBF (to see any microcytes)
- Serum iron profile

(Iron therapy should be given if deficient, before venesection.)

Q. Causes of death in TOF – (Nazrul sir)

- **Cyanotic spell** – progressive ↑ in rate & depth of respiration culminating in paroxysmal hyperapnoea, deepening of cyanosis, limpness, syncope (T-LOC), occasionally convulsions, CVA or death. (Peak incidence 6 months to 2 years)
- Thrombotic (e.g. Cerebral thrombosis, cerebral venous sinus thrombosis) & thrombo-embolic manifestations
- Brain abscess (more common after 2 years)
- Infective endocarditis
- Occasionally heart failure

Q. Management of Eisenmenger syndrome – (Gaffar sir)

- High flow oxygen (Chronic nocturnal O₂ therapy – not beneficial)
- IV fluid
- IV antibiotic
- IV Sodibicarb
- IV Morphine
- **IV Nor-adrenaline**

NO DIURETICS

Medical management of Eisenmenger's syndrome:

- **Anticoagulation** – controversial (can predispose to haemorrhage or haemoptysis), helpful in preventing thromboembolic event
- **For symptomatic hyperviscosity** – phlebotomy with isovolemic replenishment
- **Routine phlebotomy is contraindicated** because of its effect on iron stores, oxygen-carrying capacity, and ↑risk of stroke
- Management of right-sided HF is problematic, and use of digoxin in these patients is controversial. **Diuretics should be used cautiously because aggressive diuresis predisposes to hyperviscosity and decrease preload.**
- **Endocarditis prophylaxis** is warranted. (Griffin 5e, p.475)
- **Pulmonary vasodilators** – ETA, prostacyclin analogs, PDEi. – safe & likely beneficial

Surgical management of Eisenmenger's syndrome:

- Combined heart-lung transplantation or lung transplantation with concomitant repair of the intracardiac defect, if feasible

Eisenmenger's syndrome in special situations:

- Travel to areas of high altitude should be avoided. Air travel is not contraindicated.
- Pregnancy is generally contraindicated. Contraceptive methods (without estrogen).
- Noncardiac surgery – high-risk

Q. TOF – biochemistry

- Hb – increases
- Uric acid – increased (No Low-dose aspirin)

- Haematocrit – increased
- Platelet – may be decreased (If low, no anti-platelet)
Thrombocytopenia/ thrombocythaemia
- Potassium – Upper normal limit (due to secondary polycythaemia)
- Coagulation factor – vWF – decreased

Q. TOF with good sized pulmonary artery –

- Pink tetralogy (1/3 cases have mild pulmonary valvular obstruction with large VSD)
- PDA
- MAPCA

Q. DORV (Double Outlet Right Ventricle) –

- Cyanotic congenital heart disease
- Both great arteries arise entirely or predominantly from a well developed RV
- Invariably a well developed LV is also present
- **Types:**
 - Subaortic VSD without/with PS
 - Subpulmonic VSD without PS (Taussing-Bing anomaly)
 - Doubly committed VSD
 - Non-committed VSD
 - Intact ventricular septum/ closed VSD
- **Presentation:**
 - Large VSD without PS and low pulmonary vascular resistance – Mildly cyanotic infants with cardiomegaly, CHF, loud P2 and PSM over LPSB.
 - TOF like presentation (Presence of VSD and PS)
 - Taussing-Bing anomaly (Non-restrictive subpulmonic VSD with transposition like physiology) (Associations - CoA, PDA, subaortic stenosis)
 - Non-restrictive VSD like presentation (No cyanosis initially)
 - In later stage, Eisenmenger syndrome

Q. A patient with Pulmonary atresia + VSD + MAPCA – how survived?

Oxygenation through MAPCA.

Q. Cor triatrium – (Satpathy)

A membranous diaphragm dividing LA into two chambers,

- proximal chamber (PC) – accept the pulmonary veins
- distal chamber (DC) – communicate with MV & LAA. Fossa ovalis also here.
- **Cor triatrium sinister:** Partition inside LA.
- **Cor triatrium dexter:** Partition inside RA. (Extremely rare type)

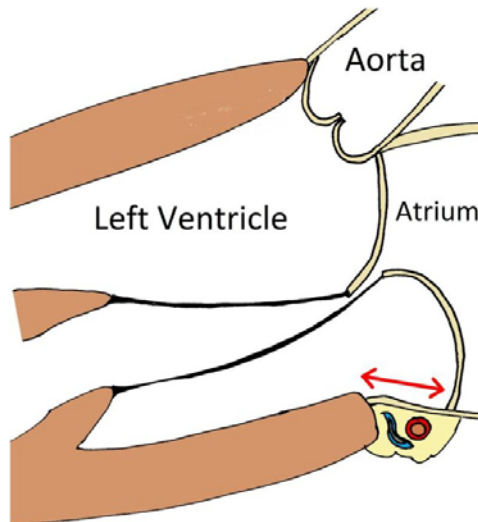
Haemodynamics: The physiologic consequence of cor triatrium are directly related to the size of the orifice between the pulmonary venous chamber and distal LA.

C/F: related to severity of lesion, features of **pulmonary hypertension**.

Echo: membrane in LA dividing into PC & DC. Colour Doppler shows turbulence across the membrane.

Q. Mitral annular disjunction (MAD) in Echo: (Aziz bhai)

- Abnormal atrial displacement of the mitral valve leaflet hinge point.
- Associated with MVD & ventricular arrhythmias, SCD.



Q. Parachute mitral valve → Congenital MS (Satpathy) Nazrul sir

- Insertion of all the variable fused chordate tendinae into a single papillary muscle group (One papillary muscle is normally absent)
- Valve tissue becomes funnel like.
- Commissures are invariably absent.
- Orifice almost eccentric, located above the papillary muscle
- This parachute MV may be incompetent → **MR**

Q. Bicuspid aortic valve (BAV) – can cause valvular AS

- BAV – **most common congenital heart defect**
- Prevalence: 0.5-2% (Griffin, 5e); M:F=3:1; 10% of first-degree relatives
- Most commonly as a result of fusion of RCC & LCC (80%) or fusion of RCC & NCC (20%)
- Calcification only in the late stage
- **Closure line of the AV in diastole is eccentric** or off-centered closer to one of the two aortic walls (normally AV closure line is equidistant)
- Concurrent dilatation of thoracic aorta occurs as many as 50% of patients, while CoA, aortic dissection and coronary anomalies are seen in a minority of patients.
- ? % of AS

BAV – (NHF-2018)

- Also evaluate aorta (4 dimensions), and Arch of aorta
- If valve requires surgery, aorta replacement if > 45mm
(If BAV + aorta >40mm, yearly F/U of aorta size; surgery if >50mm or increase by >5mm/year)
(Griffin)

Q. Frusemide dose / Lasix dose in Acute LVF –

1 ample =20 mg/ 2 ml

Dose: 0.1-0.4 mg/Kg/hr (Gaffar sir)

Lowest dose for **60 Kg** adult $60 \times 0.1 = 6$ mg per hour (if 0.1 mg/Kg/hr)

In syringe pump: (50 cc syringe attached with PMO line connected to IV cannula)
(PMO line = Pressure monitoring line, 200cm)

- 40 ml Normal saline + 5 amp Lasix (10 ml) = 50 ml
- **50 ml = 100 mg frusemide**
- **START WITH 3 ml/hr** (if 0.1 mg/Kg/hr)

60 Kg x 0.1mg/Kg/hr = 6 mg = 3 ml/hour,

60 Kg x 0.2mg/Kg/hr = 12 mg = 6 ml/hour,

60 Kg x 0.3mg/Kg/hr = 18 mg = 9 ml/hour,

60 Kg x 0.4mg/Kg/hr = 24 mg = 12 ml/hour,

- Bioavailability of oral frusemide: 50% (Wadud sir)

Oral 40 mg (Tab. Lasix 40mg) = 1 amp (20 mg) IV

Q. NA dose/ Noradrenaline dose/ NorAd dose –

(1 amp. = 2 mg/ 2ml), (2 amp + 100 ml NS)

Dose 2-30 µg/min

IV @8 µd/min (5 µg/min)

IV @15 µd/min (10 µg/min)

IV @30 µd/min (20 µg/min)

Q. Dose of IV Labetalol in hypertensive emergency – LABETALOL DOSE

- Can be used in pregnancy
- 1 amp = 50 mg/ 10 ml (Similar to GTN)
- **Bolus dose: 0.25-0.5 mg/kg** (ESC 2018 Arterial Hypertension)
4 ml (20 mg) stat, after 10 min 4 ml (20 mg),....max 300 mg
- **Maintenance dose:** 1-2 mg/min (Chinmoy);
2-4 mg/min until goal BP is reached, thereafter 5–20 mg/h
(ESC 2018 Arterial hypertension)
- 4 amp (40ml) + 60 ml NS, 100ml= 200mg, 1ml= 2mg
- **1 mg/min, 0.5 ml/min, 8d/min, 32 µd/min**
- In syringe pump, 5 amp = 50ml=250mg, 1ml=5mg
For 1-2mg/min, 60-120mg/hr, 12-24ml/hr
- Switching to oral drug, Tab. Labetalol 200 mg, 1+0+1 or 1+1+1

Q. Dose of GTN in hypertensive emergency/ GTN dose: 5-200 µg/min

- **Inj. GTN 50 mg, 1 amp + 40 ml NS or 5% D/A in syringe pump @ 0.3-1.2 ml/hr**
- 1 amp = 50 mg/ 10ml (Similar to Labetalol)
- 50 ml= 50mg, 1ml= 1mg = 1000µg
- **5µg/min** = 5µg x 60min = 300µg/ hr = **0.3 ml/hr**
- 10µg/min = 10µg x 60min = 600µg/ hr = 0.6 ml/hr
- 15µg/min = 15µg x 60min = 900µg/ hr = 0.9 ml/hr
- 20µg/min = 20µg x 60min = 1200µg/ hr = 1.2 ml/hr

- Start with 0.3 ml/hr, **increment dose every 10 min** (0.6, 0.9, 1.2, 1.5, 1.8, 2.1, 2.6, 3.1...ml/hr), with BP monitoring

Q. Classify hypertension in pregnancy – ESC 2018 Arterial hypertension (Gaffar sir)

- **Pre-existing hypertension:** precedes pregnancy or develops before 20 weeks of gestation, and usually persists for more than 6 weeks post-partum.
- **Gestational hypertension:** develops after 20 weeks of gestation and usually resolves within 6 weeks post-partum.
- **Pre-existing hypertension plus superimposed gestational hypertension with proteinuria.**
- **Pre-eclampsia:** Gestational hypertension with significant proteinuria (>0.3 g/24 h or ≥ 30 mg/mmol ACR).
- **Antenatally unclassifiable hypertension:** when BP is first recorded after 20 weeks of gestation and it is unclear if hypertension was pre-existing. Reassessment 6 weeks post-partum will help distinguish pre-existing from gestational hypertension.

Q. Clonidine as antihypertensive – (Catapres/ Clonipres)

- Central adrenergic inhibitor (central α_2 receptors)
- Dose: 0.1-0.8 mg daily
- Drug intolerance, clonidine rebound, serious autoimmune reaction

Q. MS with Hypertension (or Hypertension with MS)

Target HR 70/min

- β -blockers
- Diuretics

Q. HOCM/ AS with Hypertension (or Hypertension with AS/ HOCM) –

Target HR 70/min

- **β -blockers**
- (Cautiously Low-dose diuretic/ Vasodilators)

Q. MR with Hypertension or AR with Hypertension –

Hypertension with MR or Hypertension with AR –

Treat only if SBP > 140 -150 mmHg as ISH, to keep HR in upper normal limit

- Diuretics
- CCB – Preferably Nifedipine, alternative Amlodipine (If good LV function)
- ACEi/ ARB (If LV dysfunction)

Target HR in upper normal limit – to decrease diastolic time, decreases regurgitation

Vasodilators – causes peripheral pooling of blood

Q. MR with Hypertension or Hypertension with MR –

- Diuretics
- CCB (If good LV function)
- ACEi/ ARB (If LV dysfunction)

Q. Which drugs reduce central pressure (aortic pressure) at night –

CCB (Amlodipine), ACEi/ARB

Q. Role in ACEi/ ARB in MR –

ACEi/ ARB increases flow through physiological channel (AV) and
Decreases flow through pathological channel (MV).

Q. Which drugs reduce peripheral pressure –

β-blockers, diltiazem, Verapamil

Q. Bifascicular block – Types (Gaffar sir)

- Adjacent: RBBB + LAD (Left anterior hemiblock, LAHB)
- Non-adjacent: RBBB + RAD (Left posterior hemiblock, LPHB)

Left posterior fascicle has dual blood supply (LAD + PDA). So, LPHB is rare.
If LPHB occurs, there is more extensive damage.

- Non-adjacent is more dangerous as more tissue is damaged, may lead to CHB.
- Non-adjacent bifascicular block complicating acute MI – may need TPM.

Q. ECG features of trifascicular block – (Gaffar sir)

1. RBBB + RAD/LAD + First degree HB/ Mobitz type I or II Second-degree HB
2. LBBB + First degree HB
3. Alternate LBBB – RBBB (In monitor or ECG)

Q. RBBB – slurring of S wave in V₅₋₆, I, aVL (Gaffar sir)

LBBB – slurring of S wave in V₁₋₂

Q. T wave after tachyarrhythmia reversion (i.e. SVT reversion) – (Wadud sir)

- Anterior T wave → Memory T wave
- After reversion of SVT, ECG may transiently show the features of Left main disease/ TVD (ST elevation in aVR and ST depression with T inversion in other eight leads)

Q. Critical lesions in coronary arteries –

Left main if 50% or more stenosis

Others – 70% or more stenosis

Q. Sinus node dysfunction – (Gaffar sir)

- Sinus bradycardia
- Sinus pause/ arrest – (problem with P cell/pacemaker cell, which is centrally located in the SA node) PP interval, not exact multiple
- **Exit** block – (problem with T cell/ transitional cell, which is peripherally located in the SA node. T cell conducts impulse via 3 internodal pathways to AV node) – PP interval (not RR), **ex**act multiple
- **Sick sinus syndrome (Tachy-brady syndrome)** – **Atrial tachycardia** with various forms of SA node dysfunction
- Chronotropic incompetence – (ETT is done to confirm)
- ? Chronic AF – SA node

NB. Sinus tachycardia is not SA node dysfunction.

(Sick sinus syndrome is tachy-brady syndrome, a part of sinus node dysfunction.) (Wadud sir)

Q. Sinus tachycardia – (Griffin 5e, p.307) (Khaleq sir)

- >100/min,
- may be as high as 200/min in young, 150/min in older.

Q. Inappropriate sinus tachycardia – (Griffin 5e, p.307) (Gaffar sir)

1. HR >100/min

(Very often, rate of the tachycardia is inappropriately high at rest or during physical activity)

2. P wave axis & morphology during tachycardia similar to that during sinus rhythm
3. Exclusion of secondary causes of sinus tachycardia (No definite causes can be identified.)
4. *Exclusion of atrial tachycardia*

5. Symptoms clearly documented to be related to resting or easily provoked sinus tachycardia

Mechanism: Defects in vagal or sympathetic tone or intrinsic problem with the SN itself.

- Due to enhanced automaticity of the cells within Sinus node (Baltazar, p.180)

Symptoms: Palpitation, dyspnoea± chest pain

Rx: β-blockers, rate-limiting CCB; Ivabradine, Catheter ablation in medically refractory cases

Q. Define sinus bradycardia – (Wadud sir)

- Sinus rate <60/min
- Every QRS is preceded by P wave
- Upright P wave in lead II (inverted P in low atrial rhythm)

Q. Classify bradycardia – (Wadud sir)

- Sinus bradycardia
- **Bradyarrhythmias** –
 - o AF with slow ventricular rate
 - o Atrial flutter with slow ventricular response
 - o CHB

Q. Age-related physiological murmur – (Gaffar sir)

In elderly – MR, AR

In young – TR, PR

All stenotic murmur should be addressed.

Q. de Winter's T wave and Wellen's T wave –

De Winter's ECG pattern – an **anterior STEMI equivalent** that presents without ST segment elevation, seen in 2% of acute LAD occlusions

- **Tall, prominent symmetric T waves** in the precordial leads
- **Upsloping ST segment depression** >1mm at J point in precordial leads;
- No ST segment elevation in precordial leads;
- ST segment elevation in aVR (> 0.5-1 mm)
- Normal 'STEMI' may precede or follow the de Winter's pattern

Wellen's syndrome –

A pattern of **inverted or biphasic T waves in V₂₋₃** (in patients presenting with ischaemic chest pain) that is highly specific for critical stenosis of the LAD

Type 1 (Type B) Wellens' – deep & symmetrical T inversion

Type 2 (Type A) Wellens' – T waves are biphasic, with initial positive deflection and terminal negative deflection

Q. Causes of T inversion – (Wadud sir)

- **NSTEMI** (Deep, Symmetrical)
- **LVH with strain** (Asymmetrical, downslope shorter, upslope longer)
- Myocardial Ischaemia (Narrow based)
- HCM (Apical variety) - (Deep, Symmetrical)
- Bundle branch block
- **Hypokalaemia**, Hypocalcaemia, Hypomagnesaemia (Wide based)
- Subarachnoid haemorrhage/ Cerebral T wave (Wide based)
- Persistent **Juvenile** T wave pattern, normal finding in children (Asymmetrical)
- Wellen's syndrome
- Memory T waves (After tachyarrhythmia reversion)

Q. T wave inversion –

- **Symmetrical –**
 - Ischaemia/ **NSTEMI**,
 - HCM,
 - **Raised ICP**
 - Hypokalemia, Hypocalcaemia, Hypomagnesaemia
 - Wellen Type I
- **Asymmetrical – LVH, juvenile**
- **Biphasic T wave –**
 - Hypokalaemia (initial downsloping + terminal upsloping)
 - Wellen type II (initial upsloping + terminal downsloping)

Q. T wave in hyperkalaemia & hyperacute MI –

Hyperkalaemia: tall, symmetric, **narrow-based**, **peaked**/pointed/tented

(Proximal limb steeper than distal limb, proximal limb –rapid almost vertical ascent; distal limb, while still steep, somewhat more gradual descent & slope)

Hyperacute MI: tall, symmetric, **broad-based**, non-pointed/ not tented

Normal variant: T wave – Asymmetric, broad-based

Q. Risk assessment –

- Cardiovascular
- Mortality (TIMI, GRACE)

Cardiovascular –

- History – age, sex, menopause, OCP, smoking, DM (GTT, HbA_{1c}), Hypertension, Lipid profile, Obesity (BMI)
- In young – Thrombophilia, CRP, Ip (a)

Q. In female, after menopause hormonal protection from cardiovascular diseases for upto –

- 10 years if not diabetic,
- No protection if diabetic.

Q. Spurious hyperkalaemia – causes

- Delayed analysis
- Tight tourniquet
- Thin Needle

Q. AF – drug of choice for rate control – ABCD

- β -blockers (avoid in hypotension, bronchospasm)
- Rate-limiting CCB (Non-dihydropyridine – Verapamil, diltiazem) (avoid in hypotension)
- Digoxin (avoid in acute MI)
- Amiodarone

Q. Classify anti-arrhythmic drugs – (Vaughan Williams classification) (Opie, 8e, p.289)

- Class I – (Membrane-stabilising agent/ **Sodium channel blocker**)
 - o **Class Ia** (Block Na^+ channel and **prolong** action potential/ repolarization time) – Disopyramide, Quinidine, Procainamide
 - o **Class Ib** (Block Na^+ channel and **shorten** action potential/ repolarization time) – **(acts on ventricles only)** – Lidocaine, Mexiletine (Gaffar sir)
 - o **Class Ic** – (Block Na^+ channel with **no effect** on action potential/ repolarization time) – Propafenone HCl, Flecainide

(‘Pill in the pocket strategy’ in paroxysmal AF, 3 tab Propafenone 150 mg) (Gaffar sir)
- Class II – β -blockers (Atenolol, Bisoprolol, Metoprolol) ...**ABM**
- Class III – (Drugs whose main effect is to prolong the action potential/ markedly prolongs repolarization time) **Amiodarone, Dronedarone, Sotalol, Ibutilide, Dofetilide**
- Class IV – Diltiazem, Verapamil
- **Others – Adenosine, Digoxin (Commonly used in Bangladesh)**
- Class Ia & Ic, if used for rhythm control in AF, β -blocker or non-DHP CCB should be given.
- Propafenone (Trade name: Tab. Rytmonorm 150, 225, 300mg) – has all the side effects similar to propranolol
 - Start with 150 mg 8 hourly

Q. Amiodarone – properties

- Long half-life: 25-110 days (50 days)
- Acts on ectopic focus
- Decrease rate (β -blocking effect)
- Class III anti-arrhythmic but has all the properties of all other classes
- Used for Cardioversion
- Side effects: (Complex dose-dependant S/E)
 1. Photosensitivity, Skin discolouration
 2. Corneal deposits
 3. Thyroid dysfunction
 4. Alveolitis (Pulmonary fibrosis)
 5. Hepatotoxicity (Hepatic metabolism)
 6. Nausea, vomiting
 7. Peripheral neuropathy
 8. QT prolongation & TdP (Class Ia predispose to TdP)
 β -blockers predisposes to nodal suppression, yet give better therapeutic effects.
 9. Potentiates digoxin & warfarin

Q. Cardioversion Vs Defibrillation –

Cardioversion – Synchronous DC shock

Defib – Asynchronous DC shock

Q. CV Causes of AF –

- VHD
- Congenital heart disease (ASD)
- Ischaemic heart disease (IHD)
- Cardiomyopathy – DCM, HCM, RCM
- Pericarditis – Acute, Constrictive
- Hypertensive heart disease
- Acute massive Pulmonary embolism

5 common causes of AF – Wadud sir

- Mitral VHD
- Hypertensive heart disease
- IHD
- Thyrotoxicosis
- Lone AF

In young –

- Congenital heart disease (ASD)
- Pneumonia, septicaemia
- Myocarditis
- **Electrocution**

In 30 years/ middle age – Peripartum cardiomyopathy, Chronic RHD (MVHD)

In 40 years –

- **Chronic RHD**
- Thyrotoxicosis
- ASD +PH
- IHD/CAD
- Lone AF

In 80 years –

- ***IHD/ CAD***
- ***Hypertensive heart disease***
- ***Thyrotoxicosis***
- ***Lone AF***
- Pneumonia
- Degenerative Mitral VHD
- DCM

Complications of AF –

- Thromboembolism (2-7x↑chance)

- Heart failure (Atrial contribution in EF 20-30%) (previously EF-60%, if 20 % reduction due to AF, then 48%)
- Rx related (Warfarin)
- Dietary history – alcohol
- Pericardial effusion + brady = Myxoedema
- Pericardial effusion + tachy = Pericarditis

In ECG, clues

- LVH + strain: AS, Hypertension
- T inversion: IHD
- RVH: Chronic RHD, cor pulmonale, TOF
- small qrs in limb lead & large QRS in chest leads – Cardiomyopathy
- RBBB pattern: ASD
- Low voltage: Pericardial effusion
- Slow ventricular rate: myxoedema
- Fast ventricular rate:
- Normal ECG, low rate, T normal: Lone AF

Q. Indications of warfarin in Mitral stenosis –

- AF (Class I)
- Definite thrombus in left atrial appendage (Class I)
- Spontaneous echo contrast in LA or Giant LA (II)
- Definite H/O thromboembolism (Class I)
 - o Cardioembolic stroke (if large embolic stroke, warfarin after 1 week)
 - o MI,
 - o Mesenteric ischaemia,
 - o PAD.

Q. Classify recommendations of AHA guidelines–

Class I – must be considered

Class IIa – should be

Class IIb – may be

Class III – harm

Class IIIa – No benefit (Class III is not further classified in ESC guidelines)

Class IIIb – contraindicated or absent

Q. LVH with strain –

Asymmetric T inversion → relative ischaemia

Q. TGA & cTGA (Congenitally corrected TGA)

- TGA: Ventriculo-arterial (V-A discordance)
RV→Aorta, LV→ PA
Must have shunt (ie. PDA) for survival
- cTGA (1/13000 livebirths): **both A-V & V-A discordance**
RA→ Morphologic LV→ PA, LA→ Morphologic RV→ Aorta

No shunt necessary for survival.
Associated with complete heart block.

Q. Congenital complete heart block (CHB) –

- Presents in young age
- Less symptomatic (rate >50/min) due to Chronotropic competence (due to intact autonomic connections)
- Escape beats are reliable, arise high up, usually Bundle of His
- Responses to atropine

Causes:

- Maternal: **SLE, Rubella**
- Fetal:
 - **ASD primum**
 - Partial AV canal defects
 - **cTGA** (5-10% at birth, 10-15% by adolescent, 30% by adults)

Q. Degenerative complete heart block (CHB) –

- Chronotropic incompetence due to loss of autonomic connections
- More symptomatic
- Daytime average HR is less
- Atropine doesn't increase HR or negligible
- Escape beats arise from below (wide qrs)
- **Escape complexes unreliable**, may lead to **ASYSTOLE**
- More dangerous

Q. Stokes-Adams attacks – (Davidson)

- Sudden loss of consciousness that occurs without warning → collapse.
- A brief anoxic seizure (due to cerebral ischaemia) may occur if there is prolonged **asystole**.
- Pallor and a death-like appearance during the attack
- Characteristic flush when the heart starts beating again. there is a.
- Unlike in epilepsy, recovery is rapid.

Q. PVD(PAD): ABPI (ABI) <0.9

- If ADP is not found clinically, no need to see ABI. ADP is more sensitive than ABI. (Wadud sir)

Q. Partial AV canal defects

Q. Complete AV canal defects

Abnormal septal leaflet of TV, cleft AML of MV, ASD primum, membranous VSD

Common A-V valve

Q. Define hypertension –

- Persistent rise of blood pressure, SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg. (Wadud sir)

- The level of BP at which the benefits of treatment (either with lifestyle interventions or drugs) unequivocally outweigh the risks of treatment, as documented by trial. (ESC 2018)

Q. BP measurement – (ESC 2018 Arterial hypertension)

- Office BP,
- Out of office BP (HBPM, ABPM)
 - Nocturnal (in special situation, by ABPM)

Q. Importance of ABPM/ HBPM –

- Diagnosis of hypertension,
- Diagnosis of white coat hypertension, masked hypertension
- Response of Rx

Q. Classification of office BP – (2018 ESC Arterial Hypertension) (Mir Jamal sir)

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension (ISH)	≥140	and	<90

Q. Resistant hypertension: AHA Guidelines 2017 p133

- When a patient takes 3 antihypertensive medications with complementary mechanisms of action (a diuretic should be 1 component) but does not achieve control or
- when BP control is achieved but requires ≥4 medications.

ESC 2018 –

- When the recommended treatment strategy fails to lower office SBP and DBP values to <140 mmHg and/or <90 mmHg, respectively
- Inadequate control of BP is confirmed by ABPM or HBPM
- Adherence to therapy has been confirmed.
- The recommended treatment strategy should include **appropriate lifestyle measures** and treatment with **optimal or best-tolerated doses of three or more drugs, which should include a diuretic, typically an ACE inhibitor or an ARB, and a CCB.**
- **Pseudo-resistant hypertension and secondary causes of hypertension should also have been excluded.** (Mir Jamal sir)

Q. Refractory hypertension: (AHA Guidelines 2017)

Failure to control BP despite use of **at least 5 antihypertensive** agents of different classes, including

- a long-acting thiazide-type diuretic, such as **chlorthalidone**, and
- a mineralocorticoid receptor antagonist (MRA), such as **spironolactone**.
(**Sequential nephron block**)

Q. Hypertensive crises – Hypertensive emergencies & urgencies – (ESC 2018)

Hypertensive emergencies:

- Severe hypertension (grade 3) is associated with
- acute HMOD (hypertension-mediated organ damage) (Ongoing TOD - Wadud sir)
- often life-threatening
- requires immediate but careful intervention to lower BP, usually with iv therapy

Typical presentations of hypertensive emergency are –

- Malignant hypertension – severe hypertension (usually grade 3) associated with
 - Fundoscopic changes (flame haemorrhage ± papilloedema)
 - Microangiopathy,
 - DIC, and ... can be associated with
 - Encephalopathy (in 15% case)
 - Acute heart failure,
 - Acute deterioration in renal function.
- Acute aortic dissection, AMI, Acute LVF
- Pheochromocytoma
- Eclampsia or pre-eclampsia

Rx - over several hours, reduce MAP by 20-25% (MAP = DBP + 1/3 PP)

Immediate reduction of BP needed in following 4 conditions – (Wadud sir)

- Aortic dissection (SBP to <120 mmHg and HR <60/min)
- Acute MI (SBP <140 mmHg)
- Acute cardiogenic pulmonary oedema (SBP <140 mmHg)
- Eclampsia (<160/105 mmHg)

Q. Ten year cardiovascular risk categories (Systematic COronary Risk Evaluation system) SCORE (2018 ESC Arterial hypertension)

Very high risk – People with any of the following:

- 1) **Clinical CVD** (Acute MI, ACS, coronary or other revascularization, stroke, TIA, PAD)
- 2) **Unequivocal documented CVD on imaging** includes significant plaque (i.e. ≥50% stenosis) on angiography or ultrasound
- 3) **Diabetes mellitus with target organ damage**, e.g. proteinuria or a with a major risk factor such as grade 3 hypertension or hypercholesterolaemia
- 4) **Severe CKD** (eGFR <30 mL/min/1.73 m²)
- 5) A calculated 10 year SCORE of ≥10%

High risk –

- 1) **People with any of the following:**
 - I. **Marked elevation of a single risk factor**, particularly cholesterol >8 mmol/L (>310 mg/dL), e.g. familial hypercholesterolaemia or grade 3 hypertension (BP ≥180/110 mmHg)
 - II. **Most other people with diabetes mellitus**
- 2) **Hypertensive LVH**
- 3) **Moderate CKD** eGFR 30-59 mL/min/1.73 m²
- 4) A calculated 10 year SCORE of 5-10%

Moderate risk – People with any of the following:

- 1) A calculated 10 year SCORE of ≥ 1 to $< 5\%$
 - 2) Grade 2 hypertension
- (Many middle-aged people belong to this category)

Low risk – People with

- 1) A calculated 10 year SCORE of $< 1\%$

Q. MACE – Major Adverse Cardiac Events

Q. Signs of pulmonary hypertension – (Wadud sir)

- Epigastric pulsation
- Palpable P2
- Left parasternal heave,
- Loud P2 in pulmonary area
- **TR**
- Graham-Steel murmur (**PR** usually associated with pulmonary artery dilatation)
(Early diastolic decrescendo diastolic murmur, difficult to distinguish from AR)
- JVP – Prominent v wave due to TR, initially prominent a wave
(PH – more common in MS than MR) (Wadud sir)

Q. Pulmonary hypertension & acceleration time (PW of PV) – (Luthra-129)

PAH	AT (m/s)
Nil	> 120
Mild	100-120
Moderate	80-100
Severe	< 80

- PW of PV – early peak with mid-systolic notch
- $PAP \text{ (mmHg)} = 80 - \frac{1}{2}AT \text{ (m/s)}$
- $PAP = 4v^2 + RAP$ (v = trans-tricuspid flow velocity)

Q. Echo features of Pulmonary hypertension –

- RA, RV, PA – dilated,
 - Enlarged RV loses its triangular shape and becomes globular
 - Diameter of the PA exceeds the width of the aorta
(measure PA in midway between PA annulus and PA bifurcation) (Gaffar sir)
 - MPA, with its RPA & LPA, gives a ‘pair of trousers’ appearance.
- Small D-shaped LV during both systole and diastole (Pressure overload)
(only during diastole if due to volume overload) (Gaffar sir)
LV eccentricity index = D_2/D_1 (Gaffar sir)
- Paradoxical motion of IVS (IVS moves away from the LV and towards the RV in systole)
- PV hypertensive excursion
- TV – systolic non-coaptation
- **M-mode over PV (PSAX)** –
 - **Loss of ‘a’ dip** (flattening or loss of the normal presystolic ‘a’ wave. Due to high PA pressure, RA contraction in presystole has no effect on PV)
 - **Mid systolic notch** (due to brief closure of the PV in early systole and reopening in late systole) (**Flying W sign**) (Gaffar sir)

- **reduced EF slope**
- **Doppler:**
 - TR jet ($PASP = 4v^2 + RAP$)
 - PR jet above, >2.2 m/s; notching below
 - RVOT acceleration time: **PW of PV** – early peak with mid-systolic notch (Gaffar sir)
<80ms → Severe PH

Q. Define PAH & pulmonary venous hypertension –

- **Pulmonary arterial hypertension** is hemodynamically defined as
 - PH (i.e., $mPAP \geq 25$ mm Hg) with
 - Increased PVR (more than 3 Wood units) and
 - Normal PA wedge pressure ($PAWP < 15$ mm Hg).
 - It is a clinical condition characterized by precapillary PH and pathologic changes in the lung microcirculation.
- **Pulmonary venous hypertension –**
 $mPAP \geq 25$ mm Hg, $PVR > 3$ Wood unit, and elevated wedge pressure ($PCWP \geq 15$ mm Hg).

Q. Stages of pulmonary venous hypertension – (Gaffar sir)

- 1) Stage I – **Upper lobe diversion** ($\uparrow$ venous flow) (Normally, lower lobe diversion) (In contrast, $\uparrow$ arterial flow (PAH) – plethora)
 - 2) Stage II – Hilar prominence
 - 3) Stage III – Bat's wing appearance with
 - Kerley's A line (non-branching line, radiating from hilum, due to thick interlobal septa),
 - Kerley's B line (at lung base) (due to thick interlobar septa typically caused by **pulmonary oedema**)
 - Kerley's C line (spider web-like, throughout lung) lines
 - 4) Stage IV – Ground glass appearance (**Alveolar oedema**, clinically creps), hilar region is ill-defined or fuzzy
 - 5) Stage V – Haemosiderosis
- Radiological appearance of pulmonary oedema is due to pulmonary venous hypertension (not PAH)

Q. Pulmonary hypertension is more common in ICM than DCM. (Nazrul sir)

Q. Dana Point classification of pulmonary hypertension – (Griffin + ESC 2015)

1) Pulmonary arterial hypertension (PAH) –

- Idiopathic (IPAH)
- Heritable/ Familial PAH (Previously known as 'Primary PH')
- Drugs and toxins induced
- Association with CTD (lc-ss), portal hypertension, infections (HIV, Schistosomiasis)
- **Congenital systemic to pulmonary shunts (e.g. ASD)**

1') Pulmonary veno-occlusive disease and/or pulmonary capillary haemangiomatosis

1'') Persistent PH of newborn

2) PH due to left heart disease –

- LV systolic dysfunction

- LV diastolic dysfunction
- **Valvular heart disease**
- Congenital/ acquired left heart inflow/ outflow tract obstruction and congenital cardiomyopathies
- Congenital/ acquired pulmonary veins stenosis

3) PH due to lung disease and/or hypoxia –

- **COPD, DPLD**
- Mixed obstructive & restrictive lung disease
- Sleep-disordered breathing
- Alveolar hypoventilation syndromes
- Chronic exposure to high altitude
- Developmental lung disease

4) Chronic thromboembolic PH (& other PA obstruction)

5) PH with unclear and/or multifactorial mechanisms –

- Haematological – Chronic haemolytic anaemia, MPD, splenectomy
- Systemic – Sarcoidosis and vasculitis. Pulmonary histiocytosis, lymphangioleiomyomatosis
- Metabolic – GSD, Gaucher disease, thyroid disorders, CKD

Q. Vasoreactivity testing in PAH & CCB in pulmonary hypertension – (Gaffar sir)

Nifedipine can be given without seeing vasodilator response.

But before giving other CCB, vasoreactivity testing should be done.

Vasoreactivity testing in PAH: In PAH, vasoreactivity testing should be performed to identify patients who may benefit from long-term therapy with calcium channel blockers (CCBs).

The agent most often used to test this is

- inhaled NO, (GTN, 100% O₂ used in Cathlab) (Mohiuddin)
- IV epoprostenol.
- IV adenosine.

A positive acute response is defined as a

- **>10 mm Hg decrease in mPAP** to reach an absolute value of mPAP < 40mmHg
- Increased or unchanged cardiac output (CO) and
- Without significant drop in systemic BP.

It is important to understand the difference between **PAH vasoreactivity testing** (to select patients who may respond favourably to CCBs) and the assessment of **PH reversibility in left-sided heart failure** (for transplant).

Q. In Pulmonary hypertension, to see the prognosis, which blood test needed –

Biomarkers – Uric acid levels, Troponin T levels

- NT-Pro-BNP (Gaffar sir)
- NT-Pro-BNP below cutoff levels <1400pg/mL has been associated with better outcomes. BNP/ NT-Pro-BNP plasma levels should be checked for the initial risk stratification and may be considered for monitoring the effects of Rx.

Q. When to treat severe pulmonary hypertension without reversibility test –

- If LV function is good (Gaffar sir)
(If you treat a patient with severe pulmonary hypertension with LV dysfunction, it will worsen the condition as there is already pulmonary oedema due to LV dysfunction, decreasing pulmonary hypertension aggravate pulmonary oedema.)
- If LV dysfunction, only diltiazem can be given after cardiac cath.

Q. Plethoric & oligoemic lung fields – (Gaffar sir)

- Normally pulmonary artery is visible upto middle 1/3 (if vertically lung zones divided into – inner, middle & outer thirds), if seen upto outer 1/3 (at least 6 vessels can be traced to the outer third)→ **PLETHORIC** lung fields
- Plethoric lung fields due to ↑ pulmonary arterial flow (never venous flow)
- When hilar & intrapulmonary vessels are uniformly enlarged, it is very suggestive of **shunt lesions**.
- **Causes:** (Satpathy)
 - Acyanotic: **Shunt anomaly** (ASD, VSD, PDA), AVSD, PAPVC
 - Cyanotic (infants): dTGA, TAPVC, Single ventricle with TGA, Truncus arteriosus
 - Cyanotic (adults): TAPVC, Single ventricle with TGA, rarely Truncus arteriosus
 - Due to systemic collaterals: TAPVC (supracardiac & infradiaphragmatic types), Truncus arteriosus, tricuspid atresia, complete TGA, common atrium
- **Oligoemic lung field –** Oligoemic lung fields due to ↓ pulmonary arterial flow.
Radiologically
 - ↑ translucency
 - ↓ vascular markings (vessels are obliterated), seen upto medial 1/3
 - Pruning (=abrupt closure) (due to long standing PH, COPD)
(Pruning – rapid tapering of the peripheral arteries)
(Clinical cardiology: Current practice guidelines)
- **OLIGAEMIC** lung fields due to ↓ pulmonary arterial flow. **Causes:**
 - **Pericardial effusion**, PS, PAH
 - TOF, Eisenmenger syndrome,
- Flow is inversely proportional to pressure (in distal bed)
- If the proximal pulmonary arteries are enlarged, with pruning of the peripheral vascular markings, then PAH should be considered. (radiopaedia.org)

(Oligoemic lung field – very specific for pericardial effusion; when describing PH, better to say '↓vascular markings') (Gaffar sir)

(To confirm pericardial effusion in CXR – Trendelenburg position)

Q. VSD with plethoric lung fields means – (Gaffar sir)

- Large VSD
- Mild PH

Q. Define Eisenmenger syndrome – (Gaffar sir)

Congenital left to right shunt with shunt reversal, most probably due to severe PAH.

Q. Define Eisenmenger complex – (Gaffar sir)

VSD with shunt reversal.

Q. Moguls of the heart – the bulges of the cardio-mediastinal contour on CXR P/A view –

- **1st mogul sign** – Aortic knuckle
- **2nd mogul sign** – Pulmonary conus
- **3rd mogul sign** – LA appendage (never normal)
- **4th mogul sign** – (**LV**) bulge just above the diaphragm formed by **LV margin or cardiac apex**
- **5th mogul sign** – bulge at the **cardiophrenic angle**, may be caused by **pericardial fat pad**, pericardial cyst, or adenopathy

ASD – Cardiomegaly, Enlarged RA, RV, dilated PA with **inconspicuous aorta and its knuckle** (Jug handle type cardiac silhouette) (Satpathy)

Aortic knuckle in severe MS, ASD – narrow, inconspicuous aorta

Q. Causes of dilated coronary sinus – (Nazrul sir)

- PLSVC (Persistent left SVC)
- PS
- CCF
- Pulmonary Hypertension
- Coronary sinus type ASD

Q. Unroofed coronary sinus – (Aziz bhai)

A rare variant of ASD, where the atrial wall between the coronary sinus and LA is either partially or completely absent, resulting in a left-to-right shunt.

Type I – completely unroofed with PLSVC

Type II – completely unroofed without PLSVC

Type III – partially unroofed mid portion

Type IV – partially unroofed terminal portion

Q. Rupture sinus of Valsalva (SOV) –

Sinus of Valsalva aneurysm –

- Congenital
- Acquired (Infections - IE, TB, syphilis; atherosclerosis, injury)
- Most often involve **RCS** (?85%), less frequently NCS, rarely RCS.
- Rupture most commonly into RV, may lead to cardiac tamponade +/- intracardiac shunting

Q. In CKD, non-obstructed plaque becomes obstructed, why? (Gaffar sir)

Due to deposition of calcium (dystrophic calcification),
But hypocalcaemia is there.

Q. Patient with anaemia with bradycardia (instead of tachycardia), ECG – wide complex, Possibility

- Hyperkalaemia, most probably due to CKD (Gaffar sir)

Q. Vulnerable plaque vs stable plaque – (Gaffar sir)

	Vulnerable plaque	Stable plaque
Fibrous Cap	Thin fibrous cap	Thick fibrous cap
Cap reinforcement	Not reinforced by muscle fibre	Smooth muscle cells,

	(Few smooth muscle cells)	more/ dense extracellular matrix
Inflammatory cells	Numerous inflammatory cells infiltrates (Foam cell)	Few inflammatory cells
Lipid core	Lipid-rich	Smaller lipid pool (Poor lipid core)

Q. Anti-tachycardia pacing / Pacing in tachyarrhythmias – (Gaffar sir)

- Overdrive pacing
- Underdrive pacing
- Overdrive suppression (↑atrial rate to prevent bradycardia-induced TdP)
(Single chamber AI pacing)

Q. Myocardial stunning & myocardial hibernation –

(Reversible contractile dysfunction)

Myocardial hibernation – (Hurst 14e- 582, 958)

(Severe chronic ischaemia, ↓LV function, ↓perfusion at rest)

- Myocardium may adapt to **chronic ischaemia** by **decreasing its contractility but preserving viability**. (Dysfunctional tissue= **hibernating myocardium**.)
- *Myocardial hibernation* is a response to **chronically reduced resting myocardial blood flow**.
- Myocardium downregulates its energy expenditures for contractile work. Myocardium switches substrate selection from fatty acid to glucose.
(Anaerobic metabolism)
- Stress echo, CMR and radionuclide imaging with PET help to detect it.

Myocardial stunning – (Stunning = কিংকর্তব্যবিমূঢ়)

(Severe transient ischaemia, ↓LV function, normal perfusion at rest, large severe stress defect)

- Myocardial response to a **transient ischemic episode** caused by a **severe but transient decline in coronary flow** or an increase in cardiac work without adequate flow increase (demand-induced ischemia).
- Following the ischemic episode, **blood flow promptly recovers but contractile dysfunction of the post-ischemic myocardium initially persists** (ie, “stunning”) and recovers only gradually over subsequent hours, weeks, or months.
-
- Immediately after acute MI, surrounding ischaemic zone of infarctive zone become stunned.
- Echo – large area becomes akinetic. EF – disproportionately low.
- Improves after few days to few weeks.
- Rx – revascularization (after MPI)
- If revascularization not successful –
 - Reinfarction/ infarct extension (same vascular territory),
 - Infarct expansion

Infarct expansion (remodeling):

Acute dilatation & thinning of the area of infarction,

not explained by additional myocardial necrosis. (Gaffar sir)

- based on large infarct size and the persistence of the occlusion (Hurst 14e - 957)

- Infarct expansion occurs commonly with **anterior infarctions**, often beginning within the first 10 days, and conveys an adverse prognosis. (Hurst 14e - 404)

Disproportionate thinning & dilatation of the infarct segment probably begin within hours of acute infarction & usually reach peak extent within 7-14 days. It occurs in 35-45% of **anterior transmural MI** & to a lesser extent in infarctions at other sites. Although expansion usually develops in **large infarcts**, the extent of transmural necrosis rather than absolute infarct size predicts its occurrence. Consequences –

- Poorer exercise tolerance
- More CHF symptoms
- Greater early & late mortality
- Infarct rupture & late aneurysm formation

The risk factors associated with infarct expansion – (Hurst 14e - 968)

- cardiogenic shock,
- subendocardial infarct,
- female gender, and
- previous infarcts.

Prevention: **ACEi** should be initiated ***within the first 24 hours of onset of acute MI*** if BP allows. NSAIDs & steroids should be avoided.

Infarct extension: defined clinically as early in-hospital reinfarction after a MI. The pathologic finding is **necrotic & healing myocardium of several different recent ages** within the **same vascular territory**. Histologically more recent foci of contraction band necrosis around an infarct. (Hurst 14e- 968)

- Infarct extension results from an **incremental increase in absolute necrotic myocardium** and may be the result of infarction remote from the original infarct in either the RV or LV.
- Infarct extension usually occurs **between 2 and 10 days after infarction**, at a time when **ECG changes are evolving** and the **troponin I or T is still high**. However, the rapidly decreasing **serum creatine kinase myocardial band (CK-MB)** after the first 24 hours may be useful for the detection of infarct extension along with new Q wave on ECG.

Variables	Infarct expansion	Infarct extension
Precordial pain, changes in ECG, Haemodynamic instability	Presentation possible	
Prevalence	50-70%	20%
AMI type	Transmural	Irrespective of type
Preferred site	Anterior wall	Irrespective of wall
Infarction size	Large	Irrespective of size
Point in time	First days	Irrespective of period
Enzyme increase	No	Yes (CK-MB)

Myocardial hibernation – Hurst-965

Myocardium may adopt to chronic ischaemia by decreasing its contractility, but preserving viability. Reversibly, dysfunctional tissue is commonly referred to as hibernating myocardium. These abnormalities can be visualized by MRI, PET or ventriculography.

	Stunned myocardium	Hibernating myocardium
LV wall motion	Abnormal wall motion that tends to normalize in response to inotropes & post-extra systolic potentiation (PESP)	Abnormal wall motion that tends to normalize in response to nitrates, inotropes, PESP, PTCA or CABG
Myocardial perfusion	Adequate	Reduced
Metabolism	Adequate	Adequate

Q. Sequences of event in ischaemia – (Ischaemic cascades) (Griffin, 5e, p693)

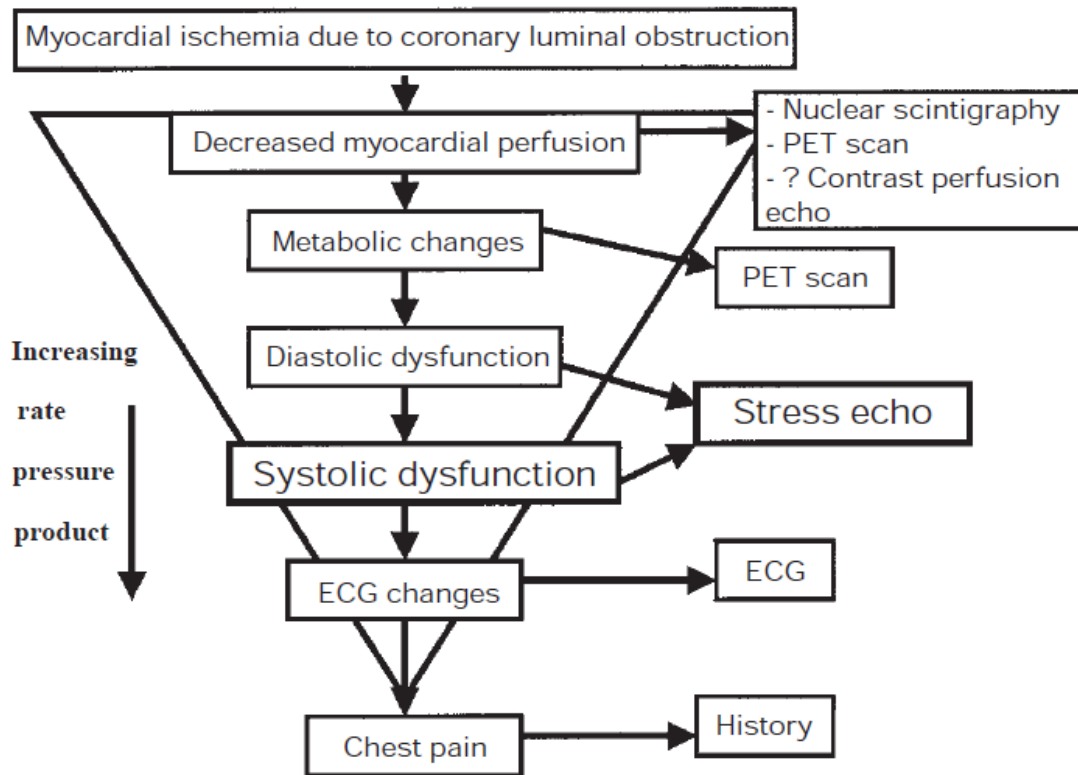
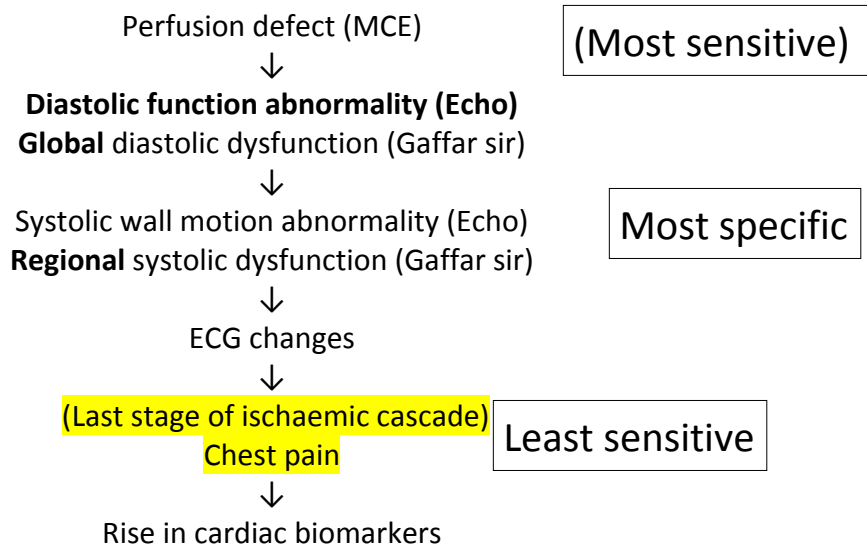


FIGURE 49.1 Ischemic cascade. PET, positron emission tomography; ECG, electrocardiogram.

Metabolic changes →



Myocardial Contrast Echocardiography (MCE)

Q. Ashman phenomenon – Baltazar

In AF, the R-R intervals are irregularly irregular. Some R-R intervals are longer and other R-R intervals are shorter. When the R-R intervals are longer or the heart rate is slower, the refractory period of the conduction tissues becomes longer. When the R-R intervals are shorter or the heart rate is faster, the refractory period is shorter. **If a long R-R interval is followed by a short R-R interval (long/short cycle)**, the atrial impulse may find the **right bundle branch still refractory** from the previous impulse and will be conducted with a wide QRS complex. This aberrantly conducted complex is the second complex (the complex with a short cycle following a long cycle). **This variability in the refractory period of the conduction system during long/short cycles in AF is called the Ashman phenomenon.**

- The **aberrantly conducted supraventricular impulse**, which is the second QRS complex after a long R-R interval. Aberrantly conducted complexes are **wide usually with a right bundle branch block pattern**. The wide QRS complex **can be easily mistaken for a premature ventricular complex. (may be mistaken as digoxin-induced)**
- Long cycle - short cycle - 3rd beat - usually RBBB morphology; (supraventricular ectopic + functional RBBB due to longer refractory period of RBB) Gaffar sir

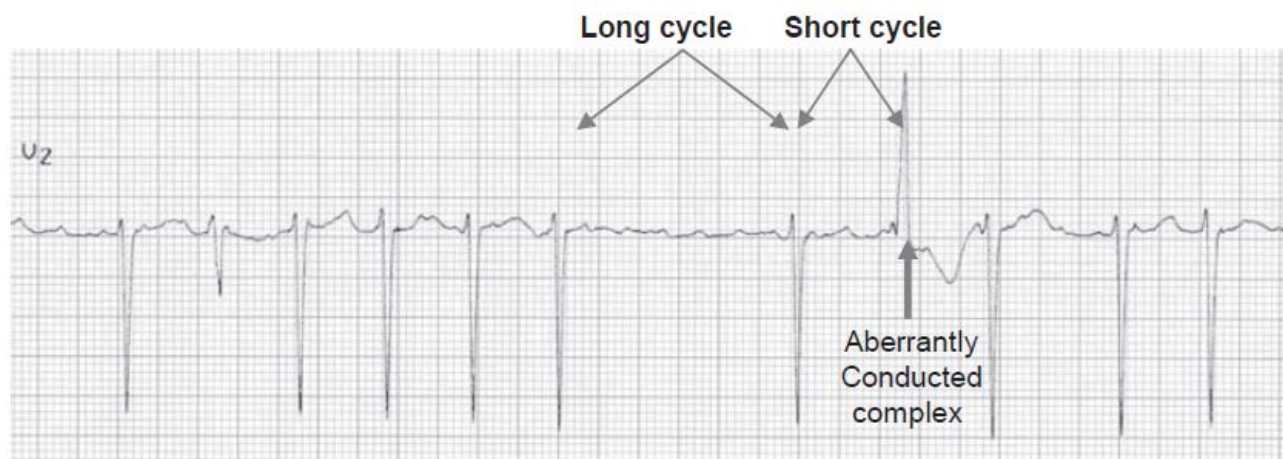


Figure 19.15: Ventricular Aberration. Arrow pointing up shows an aberrantly conducted supraventricular impulse, which is the second QRS complex after a long R-R interval. Aberrantly conducted complexes are wide usually with a right bundle branch block pattern. The wide QRS complex can be easily mistaken for a premature ventricular complex.

Q. High diastolic BP is common in – young,
In elderly, high SBP, low or normal DBP due to arteriosclerosis.

Q. Paroxysmal AF – Wadud sir

- Pill in the pocket (Propafenone, Flecainide) Dobutillide
- Risk of thromboembolism

Q. Pill in the pocket strategy for AF – (Swanton)

- For infrequent episodes of AF (previously confirmed on Holter monitoring)
- Class Ic agents (Propafenone and flecainide) – rapid onset of action compared with Amiodarone (drug working in 2 h)
- Single dose according to body weight
(Flecainide 200mg, Propafenone 450 mg if weight <70 kg;
Flecainide 300mg, Propafenone 600 mg if weight >70 kg)
- Pro-arrhythmia is rare (1%) (Atrial flutter with high ventricular rate)
- Most suitable in lone AF and infrequent episodes
- Avoided in patients with –
 - Frequent episodes of AF,
 - ECG: pre-excitation, BBB, previously documented second- or third-degree HB, long QT interval
 - Severe LV dysfunction or H/O HF
 - Severe valve disease
 - Known IHD
 - Sinusatrial disease
 - Pregnancy

Q. True dextrocardia – (Wadud sir)

- Negative complexes in Lead I
- P wave – negative in Lead I, positive in aVR
- Tall R in V₁ (Small r in V₁ in technical dextrocardia)
- 12 lead ECG in right side & assess for the ischaemia
- Investigate for risk factor profile
- Associations – Kartagener syndrome, Situs inversus, ASD, VSD, PDA

Q.

WPW syndrome – short PR, delta wave

LGL syndrome – Wadud sir

- Short PR, No delta wave
- Paroxysmal SVT, palpitation, atypical chest pain, sinister arrhythmia, SCD
- Rapid activation of ventricles, not abnormal activation,
- Nodo-fascicular pathway
- No secondary ST-T changes
- Investigations – Holter, Echo, EPS
- Rx –
 - Structurally normal heart: Reassure, β -blocker
 - Still symptomatic: EPS + ablation
 - Structurally abnormal heart – EPS + RFA

Q. Study/ Trials in cardiology –

- ARISTOTLE trial – Apixaban vs Warfarin for prevention of stroke or systemic embolism in AF (Apixaban for **R**eduction In **ST**roke and **O**ther **T**hromboembo**L**ic Events in AF)
- COURAGE trial in SCAD – Addition of PCI to optimal medical therapy does not provide any mortality benefit or improve cardiovascular outcomes. (COURAGE – Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation)

o Read Introduction & analysis

- Nobel laureate (Medicine) for propranolol (1988) – Scottish physician **James Black** (Pharmacologist)
- Angina first described by William **Heberden**
- ACCOMPLISH (**A**voiding **C**ardiovascular Events through **COM**ination Therapy in **P**atients **L**iving with **ISH**) – (Benazepril + Amlodipine) was superior to (Benazepril + HCTZ) (ISH – Isolated Systolic Hypertension)
- PLATO (**PLAT**elet inhibition & patient **O**utcome) – Ticagrelor (180mg LD, followed by 90 mg bd) vs Clopidogrel (300mg LD followed by 75mg daily) for 12 months, patients with NSTEMI-ACS & STEMI undergoing PCI
- TRITON-TIMI-38 – Prasugrel (Superior) vs Clopidogrel, ACS with scheduled PCI
- CURE (**C**lopidogrel in **U**nstable angina to prevent **R**ecurrent **E**vents) – Clopidogrel vs Placebo
- ISIS-2 (International Study of Infarct Survival) – Aspirin vs Placebo
- JUPITER (**J**ustification for the **U**se of Statins in **P**rimary prevention: An Intervention Trial **E**valuating **R**osuvastatin) trial – Rosuvastatin (long term benefit – remarkable)
- 4S – Scandinavian Simvastatin Survival Study –
- VALUE – Valsartan, Amlodipine
- ALLHAT – Lisinipril, Amlodipine
- LIFE – Losartan

Trials in HF	
Spironolactone (NYHA III-IV) vs Placebo	RALES
Bisoprolol (EF <35%, NYHA III-IV) vs Placebo	CIBIS-II
Carvedilol (EF <35%, NYHA II-IV) vs Metoprolol	COMET
Carvedilol	COPERNICUS
Digoxin (Digoxin in systolic heart failure)	DIG
Ivabradine (LVEF <40%)	BEAUTIFUL, SHIFT
Secubitril + Valsartan (ARNI) (LVEF <40%, NYHA II-IV)	PARADIGM-HF
Ramipril	HOPE
Iron deficient HF patients with IV supplementation	CONFIRM HF

Q. Which drugs (7 groups) have survival benefit/ mortality benefit – (Wadud sir)

- β -blockers
- ACEi/ ARB
- Aspirin
- Statins
- Spironolactone
- $\pm$ Ivabradine

Q. CCS (Canadian Cardiovascular Society) class of angina – (Wadud sir)

CCS class I – Ordinary physical activity does not cause angina (angina on severe exertion)

CCS class II – Slight limitation of ordinary activity (angina on moderate exertion)
CCS class III – Marked limitation of ordinary activity (angina on mild exertion)
CCS class IV – Inability to carry on any activity without discomfort (angina at rest)

- Clinical criteria; No investigations needed
- Applicable to all persons

ব্যথা শব্দটা Angina এর জন্য Appropriate না। (খারাপ/ অস্বস্তি/ চাপ লাগা)

- গ্রামের পুরুষ মানুষকে বলতে হবে, বাজারে/ মসজিদে যেতে হাপায়ে যান কিনা, খারাপ/ অস্বস্তি/ চাপ লাগে কিনা, ব্যথা হয় কিনা? (সিড়ির কথা বলা যাবেনা)
- গ্রামের মহিলাদেরকে বলতে হবে, টিউবয়েল চাপতে/ বালতি বালতি কাপড় ধুইতে পারেন কিনা/ রান্নাঘরে ভারি জিনিস সরাতে পারেন কিনা?
- সচিবালয়ের লোক - সকালে বাস ধরতে তাড়াহুড়া হলে/ ভরা পেটে/ Intercourse এর সময়/ মসজিদে দৌড়াতে খারাপ লাগে কিনা?

Q. Angina - (Wadud sir)

- Important D/D of angina – GERD
 - o Aggravates on aspirin, nitrates & CCB
 - o Nitrates & CCB – relax smooth muscle of LES
 - o Advice – Prayers before meal always, not after meal
- Risk factors – Tobacco (smoking + chewing)
- If radiating to jaw, neck/ left chest burning: suspect proximal LAD lesion
- ADP – low volume; suspect generalized atherosclerosis (No Doppler needed) Risk of CAD, stroke
- Blood tests – RBS, Lipid, Creatinine, Hb
- 50% does not have ECG changes → ETT, stress echo
 - o PLV lesion;
 - o In critical TVD, there may be balanced ischaemia, diffuse disease that develop over years)
- Exercise ECG – Treadmill test, bicycle protocol
- If contraindications of ETT – Class III angina
 - o CAG
 - o CT angiogram of coronary arteries
- Disease of left main ostium – PTCA is superior to CABG
- Disease of left main bifurcation – CABG is superior to PTCA
- PCI/PTCA in SCAD –
 - o Symptoms despite medication
 - o LV dysfunction
 - o High risk occupation

Q. Define unstable angina – (Gaffar sir)

- New-onset angina
- Angina on minimal exertion or at rest in absence of , myocardial damage
- Rapidly progressive angina/ Rapidly worsening (Crescendo)
- Not responding to sublingual nitrate
- Post-MI/ Post-PCI/ Post-CABG angina

Q. Warm-up angina/ Second wind - (Griffin, 5e, p.71) (Gaffar sir)

- Initial exertion produces angina but a similar second exertion does not reproduce angina symptoms
- During ETT, warm-up angina can be seen to be accompanied by less ST depression at similar workloads during a second exertion
- Mechanisms:
 - Collateral recruitment – Recruitment of collateral coronary flow during initial episode of ischaemia.
 - Vasodilatation of the diseased artery or subtended vascular bed, or both.
 - Ischaemic preconditioning –

Walk-through phenomenon – Resolution of angina despite continued exertion.

Decubitus angina – (less common)

- Occur with a change in posture
- Mechanism: Shift in blood volume

Nocturnal angina – Occurs at night, frequently associated with nightmares & tachyarrhythmias

First hole angina – during golf

Q. Refractory angina –

Persistence of symptoms despite optimal medical therapy and revascularization (PCI – 2 times, CABG 1 time), and no scope of further PCI. (Nazrul sir)

A chronic condition caused by clinically established reversible myocardial ischaemia in the presence of CAD, which cannot be adequately controlled by a combination of medical therapy, angioplasty or CABG.

Rx options –

- New pharmacological options
- Non-pharmacological –
 - **EECP – Enhanced external counterpulsation** (Natural bypass)
 - Neuro-stimulatory techniques –
 - Transcutaneous electrical nerve stimulation (TENS),
 - Spinal cord stimulation (SCS)
 - Angiogenesis through non-invasive techniques
 - Extracorporeal cardiac shock wave therapy
 - Angiogenesis through invasive techniques
 - **Transmyocardial laser revascularization (TMR)**
 - Percutaneous myocardial laser revascularization (PMR)
 - Stem cell/ gene therapy (preclinical/ investigational)

Q. Inj. Kebiven (USA) for parenteral nutrition –

- Macronutrients + electrolytes
- Amino acids, electrolytes, dextrose and lipids injectable emulsion.
- 3 chamber bag
- 1026 ml, 1540 ml, 2053 ml, 2566 ml

Q. Tab. Cyproheptadine – first generation antihistamine, used to ↑ appetite

Tab. Periactin 4mg (**Cyproheptadine**): Proactin (SKF), Reactin (Orion)

2 mg 6 hourly, then 4 mg 6 hourly
½ + ½ + ½ 1 week, then 1+1+1 continue

Tab. Varigestrol 160 mg, once daily – increases appetite

Q. Tab. Amiodarone 200 mg (Pacet, Cardiron, Cordarone) 100, 200 mg

1+1+1 7days

1+0+1 7 days

1+0+0 1 month

Q. Isorhythmic A-V dissociation – (Gaffar sir)

- Atrial rate = Ventricular rate
(In CHB, Atrial rate > Ventricular rate)
- Causes: Athletes, surgery, ARF, Myocarditis, IHD
- Rx: Propantheline
- Progressive shortening of PR due to SA nodal slowing
- P walk through qrs, not pass qrs

Q. Classify syncope – (Griffin, 5e, p.478, ESC 2018)

- **Reflex (neurally-mediated) syncope:**
 - o Vasovagal syncope (VVS)
 - Orthostatic – standing, less commonly after prolonged sitting
 - Emotional – fear, pain (somatic/ visceral), instrumentation, blood phobia
 - o Situational
 - Micturition
 - Swallowing, Defecation
 - Coughing, Sneezing
 - Post-exercise, post-prandial
 - o Carotid sinus syncope (1%) – cardioinhibitory, vasodepressor, mixed
- **Orthostatic hypotension (OH):**
 - o Drug-induced OH
 - o Volume depletion
 - o Neurogenic OH – Primary & secondary autonomic failure
- **Cardiac syncope:**
 - o Arrhythmia
 - Brady – Sinus node dysfunction, AV block
 - Tachy – SVT, VT, TdP
 - o Pacemaker malfunction
 - o Structural cardiac – AS, HOCM, AMI/Ischaemia, Cardiac masses, pericardial tamponade, congenital anomalies or coronary artery, prosthetic valve dysfunction
 - o Cardiopulmonary & great vessel

Classic OH: ↓SBP ≥20 mmHg & ↓DBP ≥10 mmHg within 3 min of standing

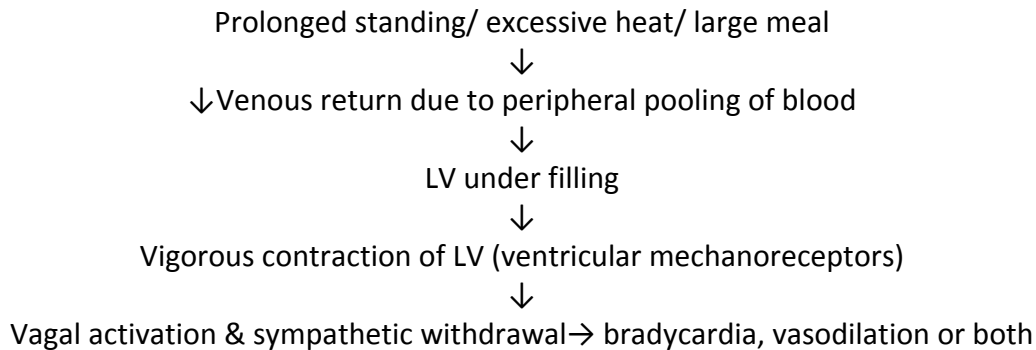
Initial OH: A brief (<30s) but exaggerated drop of in BP of >40 mmHg with rapid normalization afterwards.

Delayed OH: Slow progressive decline in BP with prolonged standing, common in elderly

Q. Vasovagal syncope (VVS) – Mechanism & Rx – (Davidson, ESC 2018 guidelines)

Causes:

- Orthostatic (Prolonged standing, less commonly prolonged sitting);
- Emotional – fear, pain (somatic/ visceral), stress,
- Instrumentation, blood phobia, hot environment

Mechanism: (Bezold-Jarisch reflex)**Provocation test:**

Head-up tilt-table test – It works via orthostatic stress induced by upright position with or without chemical stimulation (GTN, isoproterenol)

Patient supine on a padded table tilted to an angle of 60-70° for upto 45 min, while ECG and BP responses are monitored.

Indications:

- Suspected reflex syncope,
- OH,
- Postural orthostatic tachycardia syndrome (POTS),
- Psychogenic pseudosyncope (PPS)

Positive test is characterized by

- Cardio-inhibitory response: bradycardia
- Vasodepressor response: hypotension associated with typical symptoms.

Rx –

- **Lifestyle modifications**
 - Salt supplementation, (salt loading if U_{Na} excretion <170 mmol/day)
 - Avoiding prolonged standing, dehydration, missing meals
- **Midodrine** (vasoconstrictor alpha-agonist) – ‘**Pill in the pocket strategy**’ in VVS (In contrast, Chronic use of midodrine in chronic OH)
- Fludrocortisone (debatable effect),
- Disopyramide (Vagolytic & Class I_a anti-arrhythmic, block Na⁺ channel, prolong action potential),
- **β-blocker** – seldom effective & rarely used (inhibit initial sympathetic activation), (Probably unhelpful - Griffin) (Gaffar sir)
- Resistant VVS in which bradycardia is the predominant response - Dual chamber pacemaker
- Fluid

HCSS – Hypersensitive carotid sinus syndrome

MVVS – Malignant vasovagal syndrome

Rx of HCSS & MVVS: **Dual-chamber pacemaker with hysteresis**

Q. ECG changes during exercise/ ETT – (Gaffar sir)

- P peaking
- Shortening of PR
- Decrease R height
- ST depression
- Shortening of QT

Q. Exercise tolerance test (ETT) – (Gaffar sir)

- Normally – Gradual reduction of R wave height (decrease LV filling, due to ↓diastolic time as a result of ↑HR)
- Increase in QRS voltage (If R wave gradually increases) → **Ischaemic LV dilatation (Positive ETT)** (Churchill)
- Failure of BP to rise during exercise → **Ischaemic LV dysfunction (Positive ETT)** (Churchill) (Fall or failure to rise BP - Indication of termination of ETT)
- T wave changes – no value in ETT
- ST changes are important (ST depression)
- Risk of VT, cardiac arrest
- Young female with atypical chest pain – may have false positive ETT

Q. Indications of termination of ETT – (Churchill)

- Target HR achieved
- Worsening angina or excessive breathlessness
- Dizziness
- Fatigue/ patient requests test to stop
- Atrial arrhythmia other than ectopic beats
- Frequent PVCs or VT
- Worsening ST-segment **shift** (elevation or depression)
- Fall or failure to rise in BP
- Exaggerated hypertensive response to exercise (SBP>220 mmHg)
- New high-grade AV block or BBB

Q. Criteria for positive ETT – (Churchill)

- Planar ST depression ≥ 1 mm, relative to PQ segment, 80 ms after the J-point
- ST elevation
- Increase in QRS voltage (Ischaemic LV dilatation)
- Failure of BP to rise during exercise (Ischaemic LV dysfunction)
- Ventricular arrhythmias
- Typical ischaemic symptoms during exercise

Q. Sudden cardiac death (SCD) – Griffin

Death following cardiac arrest in a patient with or without known pre-existing heart disease in whom the mode and time of death are unexpected. The generally accepted timeframe between the onset of symptoms and LOC is 1 hour.

Causes:

- CAD

- Cardiomyopathies – DCM, HCM, ARVD
- Channelopathies – LQTS, Brugada syndrome, CPVT
- Others – WPW with AF,

(Structural abnormalities – HOCM, **Severe MS, AS, PS**, ICM, DCM;

Electrical abnormalities – LQTS, Brugada, ARVD) Nazrul sir

Q. After LUCS, referral/ transferred to CCU – possibilities are

- Peripartum cardiomyopathy (PPCM) – a form of DCM, where LV may not be dilated, global hypokinesia
- RHD – MS
- Congenital heart disease – ASD, VSD
- Eclampsia – (Hypertension)
- Anaemic heart failure/ fluid overload
- Acute Massive Pulmonary embolism
- Hypertensive urgency or emergency
- Aortic dissection/ MI/ Coronary artery dissection

Q. Peripartum cardiomyopathy (PPCM) –

1. Development of HF from last month of pregnancy to 5 month **postpartum**
 2. Absence of preexisting heart disease
 3. Indeterminant causes
 4. Echocardiographic features (a + b/c or a+b+c)
 - a) LV end diastolic dimension $>27 \text{ cm/m}^2$
 - b) M-mode FS $<30\%$
 - c) LVEF $<45\%$
 - Features of MR/TR, LV thrombus
- Apex – diffuse type (Not thrusting)
 - Prolonged tocolysis is a risk factor – Nifedipine, GTN, MgSO_4 , Salbutamol, NSAIDs (Indomethacin, Sulindac)
 - PPCM - Typical form develop **post-partum**, **rapid** development of symptoms (*DCM- insidious*, HF in second trimester)
 - **Pathophysiology:** excess prolactin, genetic, autoimmune
 - **Biomarkers:** NT pro-BNP (high negative predictive value), 16 kDa Prolactin
 - **Treatment:**
 - **Diuretics –**
 - **Oral thiazide** – safe during pregnancy & breastfeeding)
 - **Furosemide** can be used if the response is suboptimal
 - Do not use spironolactone to prevent hypok
 - Manage by oral potassium or diet
 - Vasodilators – use with caution, may cause uterine hypoperfusion
 - **ACEi** (Captopril/ Enalapril safe during lactation) **or ARB** after delivery (**at least ≥ 12 months**)
 - **Hydralazine & nitrates can be used safely**
 - **Benazepril, captopril, enalapril – safe in nursing women** (Tab. Enaril 5 mg)
 - MRA –
 - Spironolactone is contraindicated during pregnancy & breastfeeding,
 - Eplerenone in controversial (Pregnancy category B)

- **β -blockers – (at least ≥ 12 months)**
 - For all women with PPCM for at least 6 months after full recovery (**along with ACEi/ARB**)
 - For nursing women, **Metoprolol** is the best. (Tab. Selomet 50mg)
- **Digoxin – safe in pregnancy**
- Anticoagulation – is recommended for women with EF <35%
 - LMWH during pregnancy, warfarin postpartum.
- **Bromocriptine** (Targeted therapy) – 2.5 mg for 7 days, higher/longer Rx in severe cases (Tab. Bromodel 2.5mg stat & then, 1+0+1, 14 days)
- IVIG –
- Pentoxifylline -
- **Delivery –**
 - NVD is preferred
 - Effort to shorten second stage of labour by vacuum/forceps
 - Planned LUCS in unstable or critically ill patients
 - Combined spinal & epidural anaesthesia is preferred
- **Breastfeeding is discouraged.**
- **Prognosis**
 - 50% Full recovery (LVEF >50%) within 6 months
 - 20-25% progress to end-stage HF
 - Reported mortality 3.3 -30% over > 6 months
 - Progressive HF
 - Arrhythmias
 - Thromboembolism
 - Recovery often occurs within 2-6 months after diagnosis, but may be delayed up to 5 years
- **Poor prognostic factors:**
 - PPCM persists for > 6months
 - Poor functional status
 - LV end diastolic diameter ≥ 60 mm
 - LVEF 25-35% or less
 - Non-european ethnicity
- **Counseling for subsequent pregnancy:**
 - Chance of recurrence 30-50%
 - High risk
 - Minimum 2 year gap after recovery
 - If delayed recovery, minimum 5 year gap
 - If PPCM in subsequent pregnancy, 20% chance of death
- **Contraception:**
 - IUCD preferred
 - OCP increases the risk of thromboembolism

BOARD

B – Bromocriptine

O – Oral HF drugs
(β -blocker & ACEi after stabilization;
MRA after ablactation)

A – Anticoagulation

R – Relaxants (Vasodilators)

D – Diuretics

Q. NT pro-BNP - (NR 300-1800) (Gaffar sir)

Values increases with age.

Age <50 (NR 300-450)

Age 50-75 (NR 300-900)

- BNP positive (>400)

Q. Selenium in DCM – sources

- Nuts & legumes
- Chicken liver, neck
- Bextram Gold/Silver

Q. Valvular heart disease in pregnancy – (Long Case of F Kader bhai) AR+MR+AS

- Regurgitation is more tolerable
 - H/O ARF, OCP, previous pregnancy, family complete or not (BLTL if family complete)
 - H/O bleeding (future anticoagulation), stroke (thromboembolic), fever (IE)
 - Socioeconomic condition (solvency)
 - During pregnancy – ongoing symptomatic Rx
- Rx – Penicillin, frusemide + Potassium Syrup
- Continue pregnancy (as regurgitation lesion tolerable) with obstetric consultation
 - If NO AF or NO thrombus, NO anticoagulation
 - After pregnancy, DVR if indicated

Q. Mitral stenosis during pregnancy/ or planning for pregnancy – (Griffin 5e, p.586)

- Management depends upon the severity of stenosis, symptoms & time of diagnosis.
- If severe MS ($<1\text{cm}^2$) or moderate symptomatic MS planning for pregnancy, PTMC/ PMBV (Percutaneous mitral balloon valvuloplasty) or valve repair if PMBV is not feasible, before conception
- Mild MS ($>1.5\text{cm}^2$), pregnancy is usually tolerated with a favourable outcome.
- Optimal management of a already pregnant patient with MS is aimed at reducing heart rate and LA pressure.
 - o Selective β_1 -blocking drugs are preferred over non-selective β -blockers to avoid β_2 -mediated uterine relaxation
 - o Digoxin for AF
 - o Electrical cardioversion can be performed safely during pregnancy if the haemodynamic status warrants restoration of sinus rhythm
 - o LA pressure may be controlled by salt restriction & very judicious use of diuretics.
- PTMC during second trimester
- LUCS under G/A or epidural anaesthesia (Not Spinal anaesthesia)
- After delivery, MVR or DVR (in the same setting, aortic earlier as high pressure, mitral later)

Q. Causes of MI in pregnancy –

- Aortic dissection (during delivery),
- Thrombophilia,
- Pro-coagulant state (not should be the first answer)
(due to $\downarrow$ Protein C, $\uparrow$ stasis, venous HTN)
- Coronary thromboembolism (if associated PPCM) rarely

Weakening of the vascular walls of the medium & large muscular arteries occurs because of **estrogen-mediated decreased collagen deposition** and the effects of **circulating elastase & relaxin**. (Griffin 5e, p.581)

Q. Pregnant lady getting aspirin having normal Echo and no IHD –

Anti-phospholipid antibody syndrome

Q. Choice of anti-hypertensive drugs in pregnancy: (Gaffar sir)

- **Methyldopa** (Category B)
- **Labetalol** (Category C)
- Nifedipine SR (Category C)
- Alpha-blocker (Prazosin) (Category C) (Khalequzzaman sir)

Q. Pregnancy with hypertensive emergency – (Nazrul sir) (Gaffar sir) Mnemonic: NHL

1. **IV Labetalol** (Category C)
2. **IV GTN/ Nitroglycerine** (Category C)
3. IV Na-nitroprusside (SNP) (Category C)
4. IV Hydralazine (No longer recommended in ESC 2018) (Category C)

Q. Rational use of cardiac drugs in pregnancy –

Antihypertensives	Methyldopa B	Labetalol C	Nifedipine C	ACEi/ ARB D
	GTN C	Hydralazine C	Nitroprusside C	Prazosin C
Diuretics	Furosemide C	Bumetanide C	HCT B	Indapamide B
	Spironolactone D	Eplerenone B	Amiloride B	
Anti-platelet	Aspirin B	Clopidogrel B		
Anticoagulant	LMWH B	UFH B	Rivaroxaban NC	Warfarin D
Statins 'X'	Fenofibrate C	Gemfibrozil C		
Anti-arrhythmics	Bisoprolol C	Metoprolol C	Propranolol C	Atenolol D
	Sotalol B	Verapamil C	Diltiazem C	Amiodarone D
	Adenosine C	Digoxin C	Ivabradine NC	Flecainide C
Pulmonary vasodilators	Ambrisentan X	Bosentan X	Sildenafil B	

NC= Not classified

Statins – also contraindicated in breastfeeding

Atenolol – Hypospadias, birth defects, LBW

DC shock is a good alternative of Amiodarone. (Khalequzzaman sir)

Q. Methyldopa as antihypertensive –

(Incepta - Tab. Sardopa 250 mg, 500 mg)

- Central adrenergic inhibitor (central α_2 receptors) (like Clonidine)
- Used in pregnancy (Pregnancy category B) (Other choice – Labetalol, Nifedipine)
- Should be withdrawn immediately after delivery, because it can provoke **post-partum blue/ post-partum psychosis**
- Switch to other anti-hypertensive that are **safe in lactation**

Q. Anti-hypertensive in lactating (nursing/ breastfeeding) mother – (Gaffar sir) (6 drugs)

1. **Labetalol** (Tab. Labeta 100, 200 mg) 1+1+1
 2. Nifedipine SR (Tab. Nidipine SR 20mg) 1+0+1
 3. **Enalapril** (Tab. Enaril/Minipril 5, 10 mg) (in PPCM)
 4. **Captopril** (Tab. Acetor 25mg) (in PPCM)
 5. Metoprolol (Drug Intl -Tab. Betaloc 25, 50, XR 50, XR 100mg)
 6. Atenolol (Acme - Tab. Tenoloc 50, 100mg)
- (NEVER Methyldopa, as it may provoke post-partum blue/ psychosis)

ESC 2018 arterial hypertension –

- All antihypertensive drugs taken by the nursing mother are excreted into breast milk.
- Most are present at very low concentrations **except for propranolol and nifedipine**, with breast milk concentrations similar to those in maternal plasma.

Q. Junctional bradycardia No p wave; (on Propantheline/ Prokind 15mg + Theophylline/ Unicontin 200mg)

> Supranodal rhythm ... inverted p in inferior leads
(that means pt improved.)

Q. Left main equivalent –

- LAD ostioproximal critical >50% stenosis
- Critical TVD

Q. Features of ischaemic cardiomyopathy (ICM) –

1. RWMA (not global) – at least one wall will be thin, echogenic, akinetic
2. LVIDd >55mm
3. EF < 35%

Q. ECG changes in ICM –

- Limb leads – Tall R waves
- Chest leads – Small R waves

Q. Rx of ICM – (Nazrul sir)

- ACEi/ ARB
- β -blocker – Carvedilol
- Trimetazidine (ESC), not recommended by AHA/ACC
- Anticoagulant
- Digoxin
- Revascularization if any viable myocardium detected by MPI
- Device therapy including CRT

Q. Rheumatogenic strain of group A BHS – 1, 3, 5, 6, 18

Nephritogenic strain of BHS -

- 2, 49, 53, 55, 56, 57, 60 (Skin)
- 1, 4, 6, 12, 25 (Throat)

Q. Narrow complex tachycardia (NCT) –

- **Regular NCT –**
 - Sinus tachycardia
 - SVT (2/3 AVNRT, 1/3 AVRT)
 - Atrial flutter with 2:1 (rate 150/m) or 1:1 (rate 300/m) conduction
 - Atrial tachycardia
 - Junctional tachycardia
- **Irregular NCT –**
 - Multifocal atrial tachycardia (MAT)

- Atrial fibrillation with fast ventricular rate
- Atrial flutter with variable block
- Atrial tachycardia with variable block
- **SVT** (Baltazar)
 - Due to reentry (80-90%) – frequently seen in structurally normal heart
 - AVNRT (60%) - (Micro-reentry)
 - Typical AVNRT (slow/fast circuit)
 - 'No p wave' in 66%, pseudo S in 30%, pseudo Q in 4%
 - Atypical AVNRT (fast/slow circuit)
 - Retrograde p wave in front of QRS (RP>PR, RP<80ms)
 - AVRT (30%) – (Macro-reentry)
 - Typical AVRT (slow/fast activation)
 - Retrograde p wave after QRS (RP<PR, RP>80ms)
 - Atypical AVRT (fast/slow activation) – rare, ECG similar to atypical AVNRT
 - Retrograde p wave in front of QRS (RP>PR)
 - Concealed bypass tract (30-40%) – only retrograde (V-A)
 - Manifest bypass tract (bidirectional)
 - Orthodromic AVRT/ Narrow complex tachycardia
 - Antidromic AVRT/ Wide complex tachycardia (D/D- VT)
 - Sinoatrial reentry (SART) & Intra-atrial reentry (IART) (combinedly 10%) (Micro-reentry)
 - Due to enhanced automaticity
 - Atrial tachycardia (AT)
 - Focal AT (retrograde p wave in L_{II} in front of QRS)
 - Multifocal AT
 - Junctional tachycardia
 - Focal or paroxysmal
 - Non-paroxysmal
 - Due to triggered activity (digitalis toxicity or cardiac surgery)
 - AT with 2:1 A-V block
 - Non-paroxysmal junctional tachycardia

Q. Atrial fibrillation (AF) and atrial flutter – Mechanism

- **Atrial flutter** – **macro-reentry**, initiated by PAC
 - Unifocal atrial rhythm (Gaffar sir)
 - Cavo-tricuspid isthmus (Gaffar sir)
 - Usually arises from RA (Gaffar sir)
 - Types:
 - Type I – atrial rate 240-340/min, terminated by rapid atrial pacing
 - Typical atrial flutter –
 - Macroreentrant circuit in the **RA**, most commonly travels in a counter-clockwise rotation down the RA anterolateral free wall across the **cavotricuspid isthmus** and up the IAS
 - Atypical atrial flutter – Other macroreentrant circuits around scar tissue or surgical excision
 - Type II – atrial rate 340-440/min, NOT terminated by rapid atrial pacing

- **AF** – multiple micro-reentry
 - o Usually arises from LA (Gaffar sir)

Q. Coarse AF Versus Atrial flutter in ECG – Calipers test. (Goldberger 9e, p.151)

In AF, the atrial (f) waves vary in timing and morphology, while in atrial flutter, the atrial (F) waves are identical.

	Atrial flutter	AF
Atrial wave morphology	Identical from one F wave to another, NO BASELINE	f wave vary continuously in shape and polarity
Atrial wave timing	Identical, i.e., F-F interval “map out”.	Variable, i.e., f-f interval does not “map out”.
Atrial wave cycle length	F-F intervals ≥ 180 msec (4.5 small boxes)	Variable f-f intervals. Can be < 180 msec
Ventricular response pattern	Constant (2:1, 4:1) F/QRS ratio, or group beating pattern(s).	Completely irregular (no pattern), unless CHB or ventricular pacing is present

Coarse AF can be present with cycle lengths of 180 msec or greater.

- Coarse AF usually due to valvular cause (Khairul bhai)

Q. Pattern of atrial fibrillation – (Classify AF) (ESC Guidelines 2016)

- **First diagnosed AF:** AF that has not been diagnosed before, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms
- **Paroxysmal AF:**
 - Self-terminating, in most cases within 48 hours.
 - Some AF paroxysms may continue for up to 7 days.
 - AF episodes that are cardioverted within 7 days.
- **Persistent AF:**
 - AF that lasts longer than 7 days, including episodes that are terminated by cardioversion, either with drugs or by direct current cardioversion, after 7 days or more.
- **Long-standing persistent AF:**
 - Continuous AF lasting for ≥ 1 year when it is decided to adopt a rhythm control strategy.
- **Permanent AF:**
 - AF that is accepted by the patient (and physician).
 - Rhythm control interventions are not pursued in patients with permanent AF. (Should a rhythm control strategy be adopted, the arrhythmia would be reclassified as ‘long-standing persistent AF’.

Q. Sequelae of radiofrequency ablation of pulmonary veins in AF –

- Pulmonary venous hypertension

Q. Management of Atrial flutter – β -blocker (Gaffar sir)

- If disopyramide/ Flecainide/ Propafenone given, $\uparrow$ A-V conduction, $\downarrow$ SA node conduction; i.e. if Atrial flutter with 4:1 block (atrial rate 300/min, ventricular rate 75/min),

it will become 1:1 conduction (atrial rate 200/min, ventricular rate 200/min)

Q. Broad complex tachycardia (BCT)/ Wide complex tachycardia (WCT) – Nazrul sir

- **Regular BCT –**

- o VT
- o SVT with BBB/ aberrant conduction
- o PMT (Pacemaker-mediated tachycardia) (Gaffar sir)
- o ?Accelerated IVR
- o ?WPW with aberrant conduction (present with SVT)

- **Irregular BCT –**

- o AF with pre-excitation (WPW pattern), AF with BBB (Wadud sir)
- o TdP

- ECG - Wide complex irregular tachycardia (Wadud sir)

D/D – AF with BBB, AF with pre-excitation

Q. Which drug you will not give in AF + pre-excitation? (Wadud sir)

- Digoxin, verapamil – contraindicated.
- Amiodarone – drug of choice. Because it acts on both normal and accessory conduction pathway and both on atria & ventricles.

Q. Protocol of managing BCT/ WCT – (Gaffar sir)

- Start with (unless underlying MI or cardiomyopathy)
 - o Adenosine,
 - o Carotid sinus massage
- Then VT protocol

Q. Causes of sinus bradycardia – Rule of 2

- Raised ICP, Obstructive jaundice
- Hypothyroidism, Hypothermia
- Inferior MI, CHB, SSS
- β -blockers, rate-limiting CCB

Q. Pre-excitation & accessory pathways – (Griffin 5e, p.313)

- **Bundle of Kent** (Just below AV node, to left) – **ECG patterns**

- **Concealed accessory pathway** – inapparent by ECG (Normal ECG)
- **Manifest accessory pathway** (apparent on ECG as delta wave) -

Wolf-Parkinson-White syndrome (WPW) syndrome:

- o ECG – Short PR, slurred upstroke of QRS (delta wave), QRS >110 ms
- o Short PR due to **No AV delay**, delta wave due to **muscle to muscle slow conduction**, then goes to normal pathway (Wide QRS)
- o **Type A WPW** (Left-sided accessory pathway – dominant R wave in V1 (R Above))
- o **Type B WPW** (Right-sided accessory pathway) – dominant S in V1 (R Below)

- **AVRT with orthodromic conduction** (95%) → **NCT**

- o **Antegrade conduction occurs via AV node with retrograde conduction occurring via the accessory pathway.**
- o Rate usually 200-300/min

- o P wave may be buried in QRS or retrograde
 - o **QRS usually <120 ms** unless pre-existing BBB or rate-related aberrant conduction.
- **AVRT with antidromic conduction (5%) → BCT (WCT)**
 - o **Antegrade conduction occurs via accessory pathway with retrograde conduction occurring via the AV node.**
 - o Much less common, occurring in 5% with WPW
 - o Rate usually 200-300/min
 - o **Wide QRS** (confuse with VT, SVT with BBB) (Swanton)
- **Pre-excited AF** – (Never use digoxin)
- **Pre-excited Atrial flutter**

Rx of re-entry tachycardia in WPW syndrome – FDA (Swanton)

- **Atrial premature beat can be abolished by – Flecainide, Disopyramide, β-blockers, Amiodarone, Quinidine**
- **Antegrade conduction down the AV node is delayed by – Adenosine, β-blockers**
- **Retrograde conduction up the Kent pathway is blocked by (orthodromic) – Disopyramide, Procainamide, Ajmaline, Flecainide, Amiodarone**
- **Pre-excited AF** – (Gaffar sir)
 - o Wide QRS
 - o Rate >400/min
 - o Usually haemodynamically unstable,
 - o **No digoxin, No verapamil**
 - o very refractory to Rx (usually requires DC shock),
- **James fibre** (Just below AV node, near Bundle of His)
 - o **Lown-Ganong-Levine (LGL) syndrome**
 - o ECG – Short PR, No delta wave, normal QRS
 - o Short PR due to **No A-V delay**
 - o Normal QRS as no muscle to muscle conduction
- **Mahaim fibre** (Location: After AV node to ventricle): (Griffin 5e, p314)
 - o **Normal PR interval → AV nodal delay**
 - o Wide QRS due to muscle to muscle conduction
 - o Right-sided accessory pathways connecting either
 - AV node to ventricles (nodo-ventricular),
 - His bundle to ventricles (Hisio-ventricular),
 - Right bundle, left bundle or fascicle to ventricle (**fasciculo-ventricular**), (Griffin)
 - Atria directly to His bundle (Atrio-Hisian) (Baltazar, p.285)
 - **Atrio-fascicular** (RA→RBB) (Griffin)
 (2 most common varieties: Fasciculo-ventricular, Atrio-fascicular)
 - o Mahaim fibre tachycardia – reentrant tachycardia

Q. Cap. Recca 100 mg (**Racecadotril**, opioid agonist) 1+1+1 max 7days in AWD

Q. Poor man's exercise test –

(Textbook of Clinical Electrocardiography 3e (SN Chugh), Schamroth 7e/p181)

- **Post-PVC sinus beat – T inversion & ST depression** (Gaffar sir)

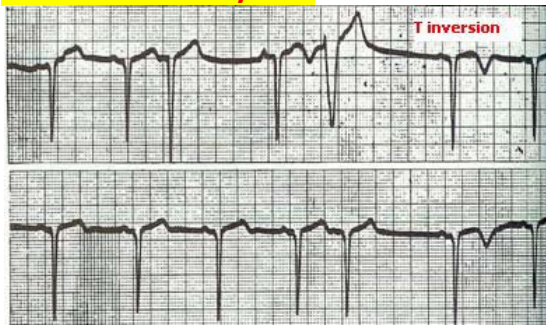
(After PVC, during compensatory pause, there will be more filling of LV. To contract overfilled LV, more exertion/stress is necessary. If ischaemic changes found in the following sinus beat, we can assume that ETT will be positive)

Post-extrasystolic T wave changes in the **first few sinus beats** following an atrial or ventricular extrasystole –

- *T waves usually becomes frankly inverted, but may at times merely show a diminution in amplitude*
- *Very rarely, inversion of U wave for one or two beats following an extrasystole carries the same significance as that of T wave.*

T wave inversion is not due to extrasystole per se, but is rather due to the pause which it produces.

Harold Levine and his associates have postulated the finding of post-extrasystolic T wave changes in the tracing recorded at rest as a '**Poor man's exercise test**', since, it indicates an immediate evidence of an abnormality and **obviates the need for a further more expensive stress test**. The abnormality consists of a **T wave change in the first sinus beat following an atrial or ventricular extrasystole**



Q. STEMI equivalents – Yahya AlThamary @facebook

- De Winter's ST/T changes
- Wellen's syndrome
- ST elevation in aVR
- New onset LBBB
- Pre-existing LBBB with Sgarbossa concordance
- True posterior MI
- Left main occlusion
- Hyperacute T wave
- T wave upright in V₁
- Isolated ST depression in aVL
- Delayed activation wave in LCX occlusion
- T wave precordial instability in LAD occlusion
- New RBBB + LAFB
- Checkmark = R on T sign

Q. Anxiolytic drugs in cardiovascular diseases –

Bromazepam (intermediate acting)

- Replace Flupentixol/Maletracin with (SSRI + BZD) Wadud sir

Q. Holmes heart – (Aziz bhai)

- Single ventricle, main ventricle is LV type (Dominant LV)
 - V-A concordance,
 - Invariably has subpulmonic stenosis
- (In 90% single ventricle with dominant LV, V-A discordance)

Q. Group beating in ECG – Causes (Gaffar sir)

- Ventricular bigeminy (first-sinus, followed by PVC)
- Atrial bigeminy
- Escape capture bigeminy (first-ectopic, then sinus)
- Mobitz type II Second-degree HB with 3:2 A-V conduction
- Mobitz type I Second-degree HB with 3:2 A-V conduction
- Rarely, Complete A-V dissociation with escape beat in couplet form (Interference dissociation)
- **Type I AV block second degree AV block** (Baltazar)
- **Sinatrial (SA) Wenckebach** (Baltazar)
(QRS complexes are grouped together because they are separated by pauses, which represent sinus impulses that are not conducted to the atrium)

Q. Cardiac cirrhosis – (Gaffar sir)

- Nutmeg liver – Centrilobular necrosis
- Stages of hepatic encephalopathy –
 - o Stage I – inversion of sleep pattern, slurring of speech, blurring of vision, fetor hepaticus, constructional apraxia
 - o Stage II – violent behaviour
 - o Stage III – pre-coma & Stage IV – coma.

Q. Causes of acute dyspnoea – (Gaffar sir)

- Cardiac – **Acute pulmonary oedema** (Acute LVF), angina equivalent
- Respiratory – Acute severe asthma, AECOPD, tension pneumothorax, Severe pneumonia, **Acute massive pulmonary embolism** (vascular cause), ARDS, inhaled FB, Lobar collapse, Laryngeal oedema (anaphylaxis), massive pleural effusion
- Metabolic – Metabolic acidosis (Mnemonic- KUSSMAUL)

	KUSSMAUL		CAT MUDPILES
K	Ketosis (DKA)	C	CO, Cyanide
U	Uraemia	A	Aminoglycosides, alcohol ketoacidosis
S	Salicylates poisoning	T	Theophylline/ Tolunene
S	(Sepsis)	M	Methanol/ Metformin
M	Methanol poisoning	U	Uraemia
A	Ethylene glycol/ poisoning (previously spelt – Aethylene)	D	DKA
U	Uraemia	P	Paracetamol metabolite (oxoproline), Propylene glycol, Paraldehyde
L	Lactic acidosis	I	Infection/ Ischaemia/ INH/Iron/IEM
		L	D-Lactate

		E	Ethylene glycol/ ethanol
		S	Salicylates/ starvation ketoacidosis

- Psychogenic – Psychogenic hyperventilation

Q. Commotio cordis – (remember the spelling) (Latin, agitation of the heart)

- In the absence of underlying cardiovascular disease, blunt non-penetrating chest blows during athletic or recreational activities that cause sudden cardiac death.
- Impact delivered directly over the heart and timing within the **vulnerable phase of repolarization** (a narrow 10-30 millisecond window just prior to the T-wave peak, equivalent to only 1-2% of the cardiac cycle) associated with peak LV pressure caused by the blow. (Hurst-1564)
- The mechanism is unclear, but it appears that a blow to the chest during an **electrically vulnerable period of cardiac repolarization** may induce VT or VF. (Griffin)

Q. PS vs PH (Pulmonary stenosis vs Pulmonary hypertension) – (Gaffar sir)

In PS – No palpable P₂, No loud P₂

In PH – palpable P₂, loud P₂

Q. Clinically D/D of PS – ASD (flow murmur in pulmonary area), AS (radiation), VSD

Q. Differentiate murmur of tricuspid regurgitation (TR) vs VSD –

TR	VSD
No thrill (as functional murmur)	Thrill
Increases during inspiration	Increases during expiration

Q. 6 'P's of acute limb ischaemia (ALI) – 2 'P' indicates acuteness

Pain	May be absent in complete acute ischaemia, and Can be present in chronic ischaemia
Pallor	
Pulselessness	
Perishing cold	Unreliable, as the ischaemic limb takes on the ambient temp.
Paraesthesia	Important features of impending irreversible ischaemia
Paralysis	

Q. Causes of bilateral absent radial pulse – (Gaffar sir)

- Takayasu arteritis
- Vasculitis
- Bilateral cervical ribs
- Anatomical aberration (radial artery in anatomical snuff box)

Q. Causes of tachycardia-induced cardiomyopathy – (Wadud sir)

- Atrial flutter
- Atrial tachycardia

Q. Blood picture following AMI – (Wadud sir)

- Neutrophilic leukocytosis
- Raised ESR & raised CRP
- Thrombocytosis

- Raised LDH

Q. From where to measure ST elevation in ECG – (Wadud sir)

- after 80 ms of J point
- J point – junction of QRS & ST segment

Q. Apex – ill-sustained & well-sustained means – (Nazrul sir)

Ill-sustained: $<1/3$ of ventricular systole

Well-sustained: $>2/3$ of ventricular systole

Q. Collaterals –

Aspirin prevents the formation of collaterals, but

Nitrate promotes the formation of collaterals.

Q. Causes of LA enlargement – (Wadud sir)

- Chronic ischaemia
- $\uparrow$ LVEDP (diastolic dysfunction e.g. LVH due to Hypertensive heart disease)
- Mitral annular calcification (MAC)

Q. In patients with aortic stenosis (AS), Echo – LV dilatation, possibilities? (Gaffar sir)

- Associated volume overload (severe AR, severe MR)
- LV systolic dysfunction

(AS causes only LV hypertrophy, NOT LV dilatation.)

Q. Cardiac emergencies –

- ACS, Acute MI, Acute LVF
- Cardiac arrest
- Aortic dissection
- Acute massive pulmonary embolism
- Hypertensive crises (Hypertensive emergencies & urgencies)
- VSR, Rupture of Sinus of Valsalva (SOV)
- Pericardial tamponade
- Prolapsing LA myxoma (Gaffar sir)

Q. Sinus tachycardia with grave diagnosis – (Gaffar sir)

- Acute massive pulmonary embolism ($S_1Q_{III}T_{III}$ only in 20%)
- Acute Aortic dissection

Q. Causes of changing murmur – (Gaffar sir)

- Prolapsing LA myxoma
- MVP
- IE

Q. Classify diuretics. (Gaffar sir)

High-ceiling/ High efficacy diuretics:

- Furosemide/ Frusemide (also causes venodilation)
- **Bumetanide** (Urinide) – Oral 0.5-2 mg/day, (1 mg Bumetanide=40 mg Frusemide)

- **Torsemide**

Medium efficacy diuretics:

- Thiazides: Hydrochlorothiazide, Bendroflumethiazide, Hydroflumethiazide
- Thiazide-like: **Chlorthalidone**, Metolazone, **Indapamide**

Weak/ adjunctive diuretics:

- Carbonic anhydrase inhibitors: Acetazolamide
- K-sparing (Aldosterone antagonist): Spironolactone, Eplerenone
- Amiloride, Triamterene
- Osmotic diuretics: Mannitol

Q. Sequential nephron blockade (SNB) – in intractable heart failure

(also in refractory hypertension + fluid retention)

- Thiazide & thiazide-like diuretics – Indapamide
- High-ceiling diuretics – Furosemide, bumetanide
- Potassium-sparing diuretics – Spironolactone

Q. Diuretic resistance – (Gaffar sir)

- To prevent diuretic resistance: Avoid single dosing for long term, Use $\frac{1}{2} + \frac{1}{2} + 0$

Q. ACE inhibitors:

Class I: Captopril-like

- **Captopril** (SH group),

Target daily dose in hypertension 25-50 mg 2x or 3x

in HF or Post-MI 50 mg 3x

Class II: Prodrugs (Carboxyl group) except Fosinopril (Phosphoryl)

- **Enalapril** (Enalaprilat) 5-20 mg in 1-2 doses (HTN), 10 mg 2x (HF)
- **Ramipril** (Ramiprilat) 2.5-10 mg in 1-2 doses (HTN), 5mg 2x (HF)
- **Perindopril**
- **Benazepril**
- **Quinapril**

Class III: Water-soluble

- Lisinopril

Enalapril:

- Longer half-life
- Slower onset of action (Enalaprilat, therapeutic effect depends on hepatic metabolism)
- Absence of SH group
- In hypertension, 2.5 mg-20 mg in 1-2 daily doses. (Twice daily better)

Q. Acute MI on Cardiogenic shock, on enoxaparin S/C –

Give IV UFH 5ml (25000 U),

1 ml (5000 IU) iv bolus, if no reaction occurs,

Remaining 4 ml (20000 IU) + 46 ml NS or 5% D/A in syringe pump iv @2.5 ml/hr (1000 IU per hour for 4 hour), gap is 4 hour in between dose.

Q. Dose of Unfractionated heparin (UFH) – 25000 Units/ 5 ml (1 ml = 5000 U)
1 ml IV stat, then (4ml + 500 ml Normal saline) 1000 U/hr
Or Inj. Heparin 250000 U + 500 ml N/S IV @5-6 drops/min (i.e. arterial thrombosis)

Q. Classify anti-platelet –

- **Cyclo-oxygenase (COX) inhibitors** – Aspirin (Irreversible inhibition)
- **Adenosine diphosphate (ADP) receptor inhibitors** – (P2Y₁₂ receptor antagonists)
 - Clopidogrel (irreversible inhibition)
 - **Prasugrel** (irreversible inhibition) (Prasugrel® 5, 10 mg)
 - **Ticagrelor (reversible inhibition)** (Ticagrelor®, Ticagrelor® 90 mg)
 - **IV Cangrelor (potent reversible inhibition)**, within < 2 min
- **Glycoprotein IIb/IIIa inhibitors – (Injectable)**
 - Abciximab (monoclonal antibody to the human GP IIb/IIIa receptor)
 - Tirofiban
 - **Eptifibatide (Integrin 10 ml vial, 2 mg/ml; 100 ml vial, 0.75mg/ml)**

Eptifibatide and Tirofiban – small molecule, reversible inhibitors.

- **Phosphodiesterase (PDE) inhibitors –**
 - **Dipyridamole** (limited use – third agent in PVD, H/O CNS bleeding)
Dipyridamole MR 200 mg twice daily if ischaemic stroke whilst on aspirin. (Davidson)
 - **Cilostazole (PDE3i)** – (also direct arterial vasodilator)

Potency: **Prasugrel** > Ticagrelor > Clopidogrel > Aspirin

Q. Anti-platelet resistance –

- May be resistant to aspirin or clopidogrel or both
- Advice: **PGx for aspirin** (2200/-) & clopidogrel (3000/-)
(done in Labaid & BIRDEM, time required: 5 days)
- Switch to Ticagrelor or Prasugrel
- Recurrent CV event/ stent restenosis, despite anti-platelet – think Anti-platelet resistance
- **PGx (Pharmacogenomics)**

Q. When anti-platelet should be stopped – (Wadud sir)

- Prasugrel – 7 days before major surgery
- Clopidogrel – 5 days before major surgery
- Ticagrelor – 3 days before major surgery
- Aspirin – 3 days before major surgery
- Cangrelor – 1 days before major surgery
- Aspirin can be continued if compelling indications (Post-PCI, device closure) in low bleeding risk surgery/ procedure (Corneal operation, endoscopy, colonoscopy, tooth scaling, skin graft etc) (Gaffar sir)
- On diagnosis of dengue, second anti-platelet (i.e. clopidogrel) should be stopped. Aspirin should also be stopped also when platelet count <100000/cmm. In case of non-PCI/CABG patients, dual anti-platelet can be stopped on diagnosis/ suspicion of dengue, if possible.

Q. Anti-platelet in stable coronary artery disease (SCAD) – (Gaffar sir)

- Single anti-platelet

- DAPT if H/O prior MI, PCI/CABG within 1 year

Q. Prasugrel – (Drugs for the heart, 8e, 2014, p368)

- Newer oral anti-platelet
- **Irreversibly** & noncompetitively inhibits the P2Y₁₂ receptor at the same site as clopidogrel
- **Prodrug**, converted to the active metabolite in liver
- Approximately 5-9 times more potent than that of clopidogrel
- **Onset of action:** within 1 hour
- **Absolute contraindications:** H/O of TIA or stroke (Griffin 5e, p.35)
- **Relative contraindications:** Age ≥75 yrs or weight <60 Kg (higher incidence of bleeding)

Q. Ticagrelor – (Drugs for the heart, 8e, 2014, p369)

- Newer oral anti-platelet
- **Reversibly** & noncompetitively inhibits the P2Y₁₂ receptor (ADP receptor)
- Plasma t_{1/2} is approximately 12 hours
- More rapid and consistent onset of action
- Additionally quicker offset of action so that recovery of platelet function is faster
- No hepatic activation required (unlike prasugrel)
- PLATO (**PLA**telet inhibition & patient **O**utcome): Ticagrelor vs Clopidogrel, NSTEMI-ACS or STEMI planned for primary PCI
- Adverse effects:
 - Increased risk of minor or non-CABG-related major bleeding
 - **Dyspnoea without bronchoconstriction** (Mechanism uncertain) (Wadud sir)
 - Within first week of Rx, transient or persist until cessation of Rx, but is not severe enough to stop Rx
 - Increased frequency of ventricular pauses ≥ 3s (Mechanism uncertain)
 - Asymptomatic increase in uric acid
- Caution is required in advanced sinoatrial disease or second- or third-degree A-V block, unless already treated by a PPM.

Q. Classify anticoagulant. (Davidson)

- **Oral anticoagulants:**
 - Vitamin K antagonist: Warfarin/ coumarins
 - Direct thrombin inhibitor: Dabigatran
 - **Factor Xa inhibitors:**
 - Rivaroxaban
 - Apixaban
 - Edoxaban
- **Injectable anticoagulants:**
 - **Antithrombin-dependent inhibitor of thrombin and Xa:**
 - Heparin
 - UFH
 - LMWH
 - Enoxaparin
 - Dalteparin
 - Tinzaparin
 - **Antithrombin-dependent inhibition of Xa:**

- Fondaparinux,
- Idraparinux
- **Direct thrombin inhibitors:** (Ideal agent in patients with HIT.) (Griffin p.37)
 - Lepirudin
 - Argatroban
 - **Bivalirudin** (Biva)

Direct thrombin (e.g. dabigatran) and factor Xa (e.g. rivaroxaban) inhibitors:

- Alternatives to warfarin.
- No blood monitoring is required.
- Fewer drug interactions.
- Fixed dosing may aid compliance.
- **Dabigatran** dose is reduced from **150 mg twice daily** to 110 mg twice daily in the over-eighties or if creatinine clearance is less than 30 mL/min.
- **Rivaroxaban** dose is reduced from **20 mg once daily** to 15 mg once daily if creatinine clearance is 30–49 mL/min, and contraindicated if below 30 mL/min.
- **Fondaparinux:** (Griffin 5e, p.16,38)
 - Heparin pentasaccharide analog that selectively inhibits factor Xa
 - Factor Xa inhibitor
 - Renally cleared, Dose: 2.5 mg S/C once daily
 - Preferred therapy in patients with increased risk of bleeding being managed with medical therapy
 - Contraindicated in patients undergoing primary PCI, because it has been associated with catheter thrombosis.
 - For patients who undergo CAG & PCI, an additional agent with anti-IIa activity (UFH or bivalirudin) should be administered given the increased rates of catheter-associated thrombosis with fondaparinux alone.
- **Bivalirudin:** (Griffin 5e)
 - Direct thrombin inhibitor
 - Reversibly inhibits thrombin
 - Short half-life of 25 min
 - Now rarely utilized in the era of radial artery catheterization given its ↑ cost.
 - 0.75 mg/Kg IV bolus, followed by 1.75 mg/Kg/h infusion. Additional boluses of 0.3 mg/Kg can be given to maintain therapeutic ACT.

Q. Mechanical prostheses and anticoagulation during pregnancy – (ESC 2018, Griffin 5e, p.269, 591) (Gaffar sir) – **UFH, S/C bolus, 1 cc tds or 2 cc bd**

- Consultation with pregnancy heart team
- It is recommended to implement changes in the anticoagulation regimen during pregnancy in hospital. (Class I)
- VKA (Warfarin) use in the first trimester results in **embryopathy** (limb defects, nasal hypoplasia) in 0.6-10% of cases. **(Fetal bone & cartilage formation abnormalities) 4-10%**
The use of warfarin through the entire course of pregnancy is associated with **'warfarin embryopathy'** is as many as **6.4%** of live births. (Griffin)
- UFH or LMWH do not cross the placenta, therefore substitution of VKA with UFH or LMWH in weeks 6-12 almost eliminates the risk of embryopathy.

- **Continuation of VKAs should be considered during the first trimester** if the **warfarin dose** required for therapeutic anticoagulation is **<5 mg/day** after patient information and consent. (Class IIa)
Risk of embryopathy is low **<3%**. (Start after careful discussion) (Griffin)
- **Discontinuation of VKAs between weeks 6 and 12**, and replacement with **adjusted-dose intravenous UFH** (aPTT $\geq 2\times$ control) or adjusted -dose LMWH (starting dose 1mg/kg for enoxaparin, subcutaneously) twice daily, should be considered in patients with a **warfarin dose >5 mg/day**. (IIa)
- In pregnant women on LMWH or UFH, it is recommended to perform **weekly anti-Xa level monitoring or aPTT monitoring** with dose adjustment (within 36 h). (Class I)
Target Anti-Xa level 0.8-1.2 U/ml 4-6h after dose. (Griffin)
Anti-Xa monitoring is essential because the therapeutic dose can increase by 50% during pregnancy. (Griffin)
- During the **second and third trimesters until the 36th week**, **VKAs** are recommended in women needing a low dose. (Class I)
- During the second and third trimesters until the 36th week, VKAs should be considered in women needing a high dose. (Class IIa)
- In pregnant women on a VKA, it is recommended to perform INR monitoring weekly or every 2 weeks. (Class I)
- During the second and third trimesters, **LMWH with anti-Xa level monitoring and dose adjustment** may be considered in women who need a high dose of VKAe after patient information and consent. (Class IIb)
- LMWH is not recommended when weekly anti-Xa level monitoring and dose-adjustment is not available. (Class III)
- It is recommended to **discontinue VKAs and start adjusted-dose intravenous UFH (aPTT $\geq 2\times$ control) or adjusted-dose LMWH at the 36th week of gestation**.
- It is recommended to **anticipate the timing of delivery** to ensure safe and effective peripartum anticoagulation. **Planned delivery** is necessary.
- If delivery starts while on a VKA or in less than 2 weeks after discontinuation of a VKA, **caesarean section** is recommended. (Class I)
Vaginal delivery while mother on VKAs is contraindicated because of the risk of foetal intracranial bleeding. Vaginal delivery requires a prior switch to IV heparin.
- It is recommended to replace LMWH with intravenous UFH (aPTT $\geq 2\times$ control) at least 36 h before planned delivery. **UFH should be continued until 4–6 h before planned delivery and restarted 4–6 h after delivery** if there are no bleeding complications.

First trimester (any one)	• Warfarin (if dose $\leq 5\text{mg/day}$) but if warfarin dose $>5\text{mg/day}$ • Dose-adjusted LMWH 12 hourly with Weekly Anti-Xa monitoring 4-6hr after the dose or • Continuous infusion of UFH (UFH, 1cc tds/ 2cc bd, s/c bolus – Gaffar sir)
Second & third trimester until 36 weeks	• Warfarin + Aspirin 75-100mg/day
36 weeks of gestation to	• Adjusted-dose intravenous UFH (aPTT $\geq 2\times$ control) or Adjusted-dose LMWH

onwards	• Replace LMWH with intravenous UFH (aPTT control) at least 36 h before planned delivery. • UFH should be continued until 4–6 h before planned delivery and restarted 4–6 h after delivery if there are no bleeding complications.
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Warfarin

- Warfarin **freely crosses the placental barrier** & can adversely affect fetal development. (UFH & LMWH do not cross placenta.)
- Associated with high incidence of
 - Spontaneous abortion,
 - prematurity,
 - stillbirth,
 - fetal bleeding.
- The incidence of **warfarin embryopathy** (fetal bone & cartilage formation abnormalities) has been estimated at 4% to 10%;
- The risks are dose-dependent, and women maintained on a dose of <5mg/day have the lowest risks.
- When administered during second & third trimesters, warfarin has been associated with
 - **fetal CNS abnormalities**, such as optic atrophy, microcephaly, intellectual disability, spasticity and hypotonia.
- Warfarin's anticoagulant effects are more potent in the fetus than in the mother because of lower fetal levels of the vitamin K-dependent clotting factors and can cause
 - **neonatal intracranial haemorrhage** or
 - **retroperitoneal haematoma**.
- Warfarin is considered **safe during breastfeeding**.

ESC 2014 – Guideline on Non-cardiac surgery: Cardiovascular assessment & Mx

- In patients with mechanical prosthetic heart valves, the evidence in favour of **intravenous UFH** is more solid; thus in some centres these patients are hospitalized and treated with UFH until four hours before surgery, and treatment with UFH is resumed after surgery until the INR is within the therapeutic range.
- On the day of the procedure, the INR should be checked. Consideration should be given to postponing the procedure if the INR is > 1.5
- LMWH or UFH is resumed at the pre-procedural dose 1–2 days after surgery, depending on the patient's haemostatic status, but at least 12 hours after the procedure.
- **VKAs should be resumed on day 1 or 2 after surgery**— depending on adequate haemostasis—with the pre-operative maintenance dose plus a boosting dose of 50% for two consecutive days; the maintenance dose should be administered thereafter.
- LMWH or UFH should be continued until the INR returns to therapeutic levels.

Target INR for mechanical prosthetic valve: (Gaffar sir)

- For MV: 2.5-3.5
- For AV: 2.0-3.0
(High target INR 3.0-4.5, AV at lower end, MV at higher end) (Opie)
- DVR or multiple valve: 3.0-4.0

- Bioprosthetic AV: Aspirin 6-12 weeks (Opie p.379)
- Bioprosthetic MV: Warfarin 6-12 weeks (Opie p.379)

Q. Bivalirudin or Eptifibatide is used during high-risk PCI - (Wadud sir)

- Left main disease
- LAD ostial lesion
- Long segment disease
- Bifurcation lesion

Q. Antidote of Rivaroxaban, apixaban – (Under trial) (Griffin)

Andexanet alfa – (Andexxa™)

A modified recombinant enzymatically inactive form of human factor Xa, which binds and reverses the anticoagulant action of the factor Xa inhibitors (e.g. Rivaroxaban, apixaban, edoxaban)

Q. Uses of diltiazem – (Gaffar sir)

(DI - Tab Cardizem 30, 60mg; SR 90, 120mg, both Tab & Cap.)

- Prinzmetal angina
- Microvascular angina

(Nitrates dilate only epicardial coronary artery)

Q. Which part of the heart is most vulnerable to ischaemia? (Gaffar sir)

- Subendocardial portion of LV, gets minimum blood supply only during diastole
- Epicardial coronary arteries gives perforating branches to myocardium

Q. Cardiorenal syndrome (CRS) – Braunwald 11e, p.1922; Hurst 14e, p.2379

- A pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other.

Five distinct syndromes have been described according to the clinical scenario and time sequence of organ failure.

- **CRS Type I (Acute Cardiorenal Syndrome):** Abrupt worsening of cardiac function (e.g. acute heart failure) leading to acute kidney injury (AKI). (Braunwald) [Predictors of CRS type I – baseline eGFR, older age, female gender, ↑baseline BP, higher initial natriuretic peptide levels & ↑CVP.]
- **CRS Type II (Chronic Cardiorenal Syndrome):** Chronic abnormalities in cardiac function (e.g. chronic heart failure) causing progressive and permanent chronic kidney disease.
- **CRS Type III (Acute Renocardiac Syndrome): (Acute worsening of kidney function leads to cardiac injury and/or dysfunction.)** Abrupt worsening of renal function (e.g. acute kidney injury) causing acute cardiac disorder (e.g. volume overload, heart failure, Hyperkalaemia).
- **CRS Type IV (Chronic Renocardiac Syndrome): (CKD leads to heart injury and/or dysfunction.)** Chronic kidney disease (e.g. diabetic nephropathy) contributing to decreased cardiac function, cardiac fibrosis, or hypertrophy and/or increased risk of adverse cardiovascular events. [CKD, in particular ESRD has 3 key mechanical contributors to HF: pressure overload (related to hypertension), volume overload and cardiomyopathy.]
- **CRS Type V (Secondary Cardiorenal Syndrome):** Systemic (extrarenal & extracardiac) conditions (e.g. multiple trauma, sepsis) leading to simultaneous injury and/or dysfunction of both the heart and kidney.

Q. MINOCA (Myocardial infarction with non-obstructive coronary arteries) –

The diagnosis of MINOCA is made immediately upon CAG in a patient presenting with features consistent with an AMI as detailed by the following criteria –

- Universal AMI criteria (MI type 1 or 2)
- Non-obstructive coronary arteries on CAG, defined as **NO coronary artery stenosis >50% in any potential IRA**
- No clinically overt specific cause for the acute presentation

There are disparate aetiologies causing MINOCA and they can be grouped into:

(1) Secondary to epicardial coronary artery disorders (e.g. atherosclerotic plaque rupture, ulceration, fissuring, erosion, or coronary dissection with non-obstructive or no CAD) (MI type 1);
(2) Imbalance between oxygen supply and demand (e.g. coronary artery spasm and coronary embolism) (MI type 2);

(3) Coronary endothelial dysfunction (e.g. microvascular spasm) (MI type 2); and

(4) Secondary to myocardial disorders without involvement of the coronary arteries (e.g. myocarditis or Takotsubo syndrome). [(3) & (4) **Myocardial injury conditions**]

- Performance of **CMR** within 2 weeks after onset of symptoms should be considered to increase the diagnostic accuracy of the test for identifying the aetiological cause of MINOCA. (ESC 2017 STEMI guideline)

Q. Kounis syndrome/ Allergic ACS – (FCPS Jan 2019)

- Acute coronary syndrome caused by anaphylaxis
- Mast cell activation and release of cytokines leads to coronary spasm or plaque rupture
- Types:
 - Type I: Patients with no underlying CAD
 - Type II: Patients with underlying asymptomatic CAD
 - Type III: Eosinophils/ mast cells in coronary or stent thrombosis
- Management:
 - Treat anaphylaxis with adrenaline (be cautious for coronary spasm)
 - Corticosteroids, antihistamine, vasodilators, mast cell stabilizers
 - β -blockers are contraindicated.
 - Avoid morphine (may induce mast cell degranulation)

Q. ALCAPA – Anomalous Left Coronary Artery arising from the Pulmonary Artery (BD echo-2018)

Q. Causes of CAD – (Gaffar sir)

- **Atherosclerosis**
- **Vasospasm**
- **Athero-thrombosis**
- **Coronary embolism**
- Coronary artery dissection, Coronary artery anomalies
- Vasculitis/ inflammation

Q. Spontaneous coronary artery dissection (SCAD) – (Gaffar sir)

Risk factors:

- Young female, Women in 40s or 50s (F>M)
- Post-delivery
- Coronary artery dysplasia (Fibromuscular dysplasia, FMD)
- Unaccustomed sudden strain, extreme physical exercise

- Extreme emotional stress
- SLE, PAN
- Inherited CTDs – EDS, Marfan's syndrome
- Uncontrolled hypertension
- Cocaine or other drug abuse

Q. Classify coronary/ ischaemic heart disease (IHD) – (Wadud sir)

- Chronic CHD –
 - o Stable CAD (SCAD),
 - o ICM
- **Chronic coronary syndrome (CCS)** – (New term) (ESC 2019)
 1. Patients with **suspected CAD** and 'stable' angina symptoms, and/or dyspnoea
 2. Patients with **suspected CAD** and new onset of HF or LV dysfunction
 3. Asymptomatic and symptomatic patients with stabilized symptoms **<1 year** after an ACS, or patients with recent revascularization
 4. Asymptomatic and symptomatic patients **>1 year** after initial diagnosis or revascularization
 5. Patients with angina and **suspected vasospastic or microvascular disease**
 6. Asymptomatic subjects in whom CAD is detected at **screening**
- Acute coronary syndrome (ACS) –
 - o UA,
 - o NSTEMI,
 - o STEMI
 - o Sudden cardiac death (SCD)
- HF (Amal sir)

Q. Causes of ST elevation other than Acute STEMI – (Baltazar)

- Ventricular aneurysm (No reciprocal changes) (Wadud sir)
- Acute Pericarditis (ST-T ratio in V6 is >25%)
- Brugada ECG
- Early repolarization
- LBBB, LVH, LV aneurysm
- WPW syndrome
- Osborn wave of hypothermia
- Normal elevation at transition zone
- Hyperkalaemia, Hypercalcaemia
- Others – Pacemaker rhythm, Ventricular ectopic, Takotsubo cardiomyopathy, tumors & trauma involving ventricle

Acute STEMI – have reciprocal changes. (Wadud sir)

Inferior MI – reciprocal changes in I, aVL.

Q. Causes of ST elevation in V1 –

- Acute MI anteroseptal
- RVI
- ?Left main disease
- Hyperkalaemia (**dialyzable current of injury**)

- Early repolarization
- Brugada pattern

Q. Inverted U wave signifies – Ischaemia, volume overload

Q. Post-MI – Nitrate 6 weeks

- **Acute MI with stroke** – Nitrate cautiously

Q. Haemorrhagic stroke after STK –

- Anti-platelet after 2 weeks

Q. Haemorrhagic stroke presents with AMI later –

- Anti-platelet after 2 weeks
- LMWH after 3 months of haemorrhagic stroke
- Never STK after haemorrhagic stroke any time in the past

Q. Acute STEMI inferior with recurrent VT – Echo – Pseudoaneurysm

Q. Pseudoaneurysm & true aneurysm in Echo –

Pseudo-aneurysm – (Luthra)

- Narrow neck,
- posterolateral LV wall,
- expansile (**thin wall**, expand in systole),
- wall – pericardium,
- **friable & liable to rupture**,
- **(multilayered) thrombus** (clotted haemopericardium) fills cavity.

(Because the **risk of rupture is high**, accurate diagnosis and prompt surgical repair of pseudoaneurysms is important.)

LV true aneurysm – (Luthra) (Hurst 14e – 404)

- **wide neck**, (wide mouthed, **thin-walled** myocardial segments)
- at or near LV apex,
- **dyskinetic expansion** during systole,
- wall – myocardium – more echogenic,
- rupture unlikely,
- often associated with pedunculated or laminar thrombus;
- more common after **anterior MI**.

Q. Dyskinesia – Systolic thinning and outward bulging

Q. Aneurysm – permanent dilatation

Q. Ectasia & aneurysm – (Wadud sir)

- **Ectasia** – vessel diameter ↑ upto 1.5 fold
- **Aneurysm** – vessel diameter ↑ more than 1.5 fold

Q. A patient present with persistent ST elevation with H/O syncope. (Atahar sir)

- LV aneurysm → VT, VF
(not stroke, because stroke usually causes focal neurological deficit)

Q. Features of RVI (RV infarction) –

- Features of MI
- Triad: Raised JVP, hypotension, clear lungs
- Features of acute MI inferior
- May present with diarrhoea (Vagal/ parasympathetic) Nazrul sir

Q. Features of Acute MI Inferior with RVI with CHB –

- Ischaemic central chest pain, lightheadedness, presyncope, syncope
- Signs –
 - Hypotension,
 - bradycardia (regular), low volume pulse (RVI)
 - JVP – raised, irregular cannon waves (CHB)/ prominent a wave (RVI)
 - Variable S1
 - Clear lungs

Clinical findings associated with RV infarction (RVI): (Griffin 5e, p.50)

Hypotension	TR
Elevated jugular venous pressure	Right-sided S3 and S4
Kussmaul sign (Failure of JVP to ↓ with inspiration)	Pulsus alternans
Abnormal JV pressure patterns (y ≥ x descent)	High-grade AV block

Q. In RV infarction (RVI), mechanical function is reversible, why – (Gaffar sir) (Griffin 5e, p.50)

Extensive, irreversible RV damage is unusual because –

- 1) RV has lower oxygen requirement because of its smaller muscle mass (wall thickness is 1/3 than that of LV),
(RV works at low workload, low pressure chamber),
- 2) RV is perfused during both systole & diastole,
- 3) RV often receives extensive left-to-right collateral blood flow.

Q. Management of RVI – (Griffin 5e, p.50) (Gaffar sir)

- Fluid administration (Fluid boluses upto a litre may be considered with monitoring of haemodynamic status). Target CVP 15 mmHg
- Inotropes – Dobutamine (then NA, Dopamine – Gaffar sir)
- Revascularization by thrombolysis or PCI
- AV sequential pacing (Longer AV delay of approx. 200 ms and HR 80-90/min) (Dual-chamber TPM) (Gaffar sir)
- IABP or temporary RV support device (RP impella/ Protek Duo)
- ?Pericardiectomy (Gaffar sir)
- Cardiac transplantation (Gaffar sir)

Q. Acute STEMI/ AMI – STEMI, ESC guidelines - 2017

8 Changes from 2012: (Mir Jamal sir)

- 1) Radial access (Class IIa → Class I)
- 2) DES over BMS (Class IIa → Class I)
- 3) Complete revascularization (Class III → Class IIa)

- 4) **Thrombus aspiration** (Class IIa → **Class III**)
- 5) Bivalirudin (Class I → Class IIa)
- 6) Enoxaparin (Class IIb → Class IIa)
- 7) Early hospital discharge (Class IIb → Class IIa)
- 8) **Oxygen when SaO₂ <90%**

Q. Management of acute STEMI – (Mir Jamal sir)

- Hospitalization, IV access, attach to monitor
- Loading dose of dual antiplatelet
(Chewable/ Soluble Aspirin 300 mg + Clopidogrel 300 mg) & Statin
- Morphine/ Pethidine + antiemetic
- Thrombolytics
- Primary PCI, if facility available
-

Q. Half-dose thrombolysis – in facilitated PCI, not done now

Q. Types of PCI – (ESC AMI – STEMI Guidelines 2017)

- **Primary PCI** – Emergent PCI without previous fibrinolytic Rx
(Emergent PCI with balloon, stent, or other approved device, performed on the IRA without previous fibrinolytic therapy)
 - It is the preferred reperfusion strategy in patients with STEMI within 12 hrs of symptom onset, provided it can be performed expeditiously (i.e. 120 min from STEMI diagnosis) by an experienced team.
- **Rescue PCI** – Emergent PCI as soon as possible in case of failed fibrinolytic Rx
- **Facilitated PCI** – Routine fibrinolysis prior to transfer for PCI (in all patients who present with STEMI results in worse outcomes) (Griffin 5e, p.21)
- **Pharmacoinvasive strategy** – Fibrinolysis combined with rescue PCI (in case of failed thrombolysis) or routine early PCI strategy (in case of successful thrombolysis) (**within 24 h**)
- Elective PCI –

Primary PCI strategy – Emergent CAG & PCI of the IRA if indicated

CHIP: Complex & high-risk indicated procedure/ patients (Mohsin sir)

DIDO: Door-in, door-out (Mohsin sir)

Q. Door-in, door-out – (ESC 2017 Acute STEMI)

- For patients presenting in a non-PCI centre, door-in to door-out time, defined as the duration between arrival of the patient at the hospital to discharge of the patient in an ambulance en route to the PCI centre,
- new clinical performance measure, and
- ≤30min is recommended to expedite reperfusion care.

Q. Important time targets in Acute STEMI –

- Maximum time from FMC (first medical contact) to ECG & diagnosis: **≤10 min**

- Maximum expected delay from **STEMI diagnosis to primary PCI (wire crossing)** to choose **primary PCI strategy** over fibrinolysis (If this target time cannot be met, consider fibrinolysis): **≤ 120 min** (Mir Jamal sir)
- **Maximum time from STEMI diagnosis to wire crossing in patients presenting at primary PCI hospitals: ≤ 60min** (Mir Jamal sir)
- **Maximum time from STEMI diagnosis to wire crossing in transferred patients: ≤ 90min**
- Maximum time from STEMI diagnosis to bolus or infusion start of fibrinolysis in patients unable to meet primary PCI target times: **≤10 min**
- Time delay from start of fibrinolysis to evaluation of its efficacy (success or failure): **60-90 min**
- Time delay from start of fibrinolysis to angiography (if fibrinolysis is successful): **2-24 hours** (Routine early PCI strategy after fibrinolysis)

FMC (First medical contact): (ESC 2017 – AMI – STEMI) (Mir Jamal sir)

- The time point when the patient is either initially assessed by a physician, paramedic, nurse or other trained EMS (Emergency medical system) personnel who can obtain and interpret the ECG, and deliver initial interventions (e.g. defibrillation).
- FMC can be either in the prehospital setting or upon patient arrival in the hospital (e.g. Emergency department)

Q. Indications of STK (streptokinase)/ Indications of thrombolysis – (Wadud sir)

1. **Ischaemic type of chest pain with**
 - **ECG – Acute STEMI,**
 - **presenting within 12 hours,** (after 12 hours, fibrin becomes organized) but can be given within 24 hours if ongoing chest pain.
 - **provided no contraindications**
 2. ECG – New onset of LBBB, with ischaemic type of chest pain
 3. True posterior MI
 4. Acute massive Pulmonary embolism with cardiogenic shock
 5. ECG - Suspected Left Main Disease/ Critical TVD (**Not agreed by Khaleq sir, No reference**) (ST elevation in aVR + ST depression in at least 8 leads)
 6. Acute limb ischaemia (ALI)
 7. Fistula block
 8. Acute right-sided prosthetic valve thrombosis (Griffin 5e, p.275) (82% success)
Left-sided prosthetic valve thrombosis with contraindication to surgery.
[82% success, associated risk of death (10%), systemic embolism (12.5%)]
Dose: 5 lac IU bolus over 20 min, followed by 1.5MU over 10h.
- **Second dose of STK** can be given
 - 1) within 5 days of first dose or
 - 2) after 6 months of previous dose. (Gaffar sir)
 - **Fibrinolytic therapy/ Thrombolytics –** in order of preference – choice of agent
 - 1) **Alteplase** (tPA) (fibrin-specific agent) (Rate of ICH: 0.7%),
Dose: IV bolus 15 mg, followed by 0.75 mg/kg (max. 50 mg) over 30 min and then 0.5 mg/kg over 60 min.
 - 2) **Reteplase** (less fibrin-specific mutation of tPA). No mortality benefit over tPA.
Dose: double bolus, 10 mg each, 30 min apart.
 - 3) **Tenecteplase (TNK):** Currently preferred agent.

- **Dose:** Single bolus. Weight-adjusted dose is 30-40 mg.
- Improved fibrin specificity, enhanced resistance to plasminogen activator inhibitor (PAI) I, decreased plasma clearance.
- Significantly less noncerebral bleeding.

4) **Streptokinase** – (Rate of ICH: 0.5%)

STREPTOKINASE:

- No longer commercially available in the US & is of historical significance.
- Overall rate of ICH is 0.5%.
- Non-fibrin-specific agent, capable of lysing circulating & clot-bound plasminogen to plasmin → substantial fibrinogenolysis, fibrinogenaemia & ↑FDPs.

Q. Contraindications of STK/ Streptokinase/ fibrinolytic therapy - from ESC Guidelines (Mir Jamal sir)

Absolute contraindications –

1. Previous **ICH** or stroke of unknown origin **at anytime** (Mir Jamal sir)
2. Ischaemic stroke in preceding 6 months
(Absolute C/I within 3 months, R/I > 3months - Griffin)
3. CNS damage or neoplasms or AVM
(Known intracranial neoplasm, structural cerebral vascular lesion or closed head injury within 3 months – Griffin 5e)
4. Recent **major trauma/ surgery/** head injury (**within preceding month**)
(Intracranial or intraspinal surgery within 2 months) (Griffin 5e)
5. **GI bleeding** with the past month
6. Known bleeding disorder (**excluding menses**) (Wadud sir)
(Active bleeding or bleeding diathesis) (Griffin 5e)
7. **Aortic dissection** (Suspected Aortic dissection – Griffin 5e)
8. Non-compressible puncture in the past 24h (Liver biopsy, LP) (Relative C/I, Griffin 5e)
9. Severe uncontrolled hypertension, unresponsive to medical therapy (Griffin 5e)
10. **For streptokinase, prior exposure within 6 months** (Griffin 5e)

Relative contraindications –

1. TIA in the preceding 6 months
2. Oral anticoagulant therapy
3. **Pregnancy** or 1 week post-partum
4. **Refractory hypertension (SBP >180 and/or DBM >110)** (Wadud sir) (Mir Jamal sir)
(Severe uncontrolled hypertension at presentation BP >180/110 mmHg, or H/O chronic severe hypertension) (Griffin 5e)
5. Advanced liver disease
6. IE
7. Active PU
8. Prolonged (>10 min) or traumatic resuscitation (Traumatic CPR) (Wadud sir)

Cardiovascular contraindications of STK – (Wadud sir)

- Aortic dissection
- Severe hypertension
- Traumatic CPR

Q. Features of successful thrombolysis – (1 hour after completion of STK)

1. Symptomatic relief of ischaemic chest pain
2. ECG –
 - I. Reduction of ST segment elevation at least 50% of highest elevation
 - II. Reperfusion arrhythmia (from starting of thrombolysis to 6h) (Wadud sir)
 1. PVC/ frequent Ventricular ectopics
 2. Junctional rhythm
 3. AIVR (Rate >40-100/m) (Wadud sir – IVR)
 4. Short runs of VTMalignant reperfusion arrhythmias: VT, VF, AF (Gaffar sir)
 - III. Early appearance of Q wave (Nazrul sir)
3. Rapid rise & fall of cardiac biomarkers (Troponin I)
4. Improvement of wall motion abnormality or EF in echocardiography
5. CAG – Improvement of TIMI flow (TIMI 3)

Q. Reperfusion arrhythmias –

1. PVC/ frequent Ventricular ectopics
2. Junctional rhythm
3. AIVR (Rate >40-100/m) (wide QRS, not preceded by p wave)
(when the ventricles exceed their intrinsic rate of 20 to 40 bpm)
4. Short runs of VT

Q. Rates in different ventricular rhythms –

Normal ventricular rate 15-40/min

- Idioventricular rhythm <40/min
- AIVR 40-100/min
- VT >100/min

Q. Define idioventricular rhythm (IVR) – 3 or more successive ventricular ectopics.

Q. Atypical presentations of IHD –

- Common in diabetics/ neuropathic patients, elderly
- Neckache, headache, toothpain
- Angina equivalent (presents with exertional SOB)

Q. Complications of acute MI – (Arrhythmias, HF, Cardiogenic shock) (Mir Jamal sir)

- Acute LVF
- Arrhythmias (almost 98% acute MI have arrhythmias) – PVC, CHB, VT, VF
- Acute Pericarditis
- Mechanical –
 - o VSR,
 - o Acute MR due to papillary muscle dysfunction (Common) / rupture
 - PMPM – PDA (single blood supply) (Griffin p.267) (Wadud sir)
 - ALPM – supplied by LAD & LCX
 - o Free wall rupture (EMD/ PEA)

Free wall rupture – Cardiac arrest (EMD/ PEA), nearly 100% mortality (Gaffar sir)

Q. Pathology of STEMI & NSTEMI – (Wadud sir)

STEMI – occlusive (non-mobile) thrombus, initially platelet-rich but finally fibrin-rich;

Less chance of rupture, Rx with fibrinolytics

NSTEMI – Non-occlusive (mobile) thrombus, platelet-rich; more chance of rupture & (? can go through blood). Rx with anticoagulants

Q. Why thrombolysis not done in NSTEMI – (Gaffar sir)

- Study shows no benefit, rather more harmful
- Non-occlusive thrombus
- Platelet-rich thrombus

Q. Define myocardial injury and MI – (2018 ESC – 4th universal definition of MI)

Myocardial injury –

- Elevated cTn values with at least one value above the 99th percentile upper reference limit (URL).
- The myocardial injury is considered acute if there is a rise and/or fall of cTn values.

Acute myocardial infarction – (Mir Jamal sir)

- **Acute myocardial injury** with
- Clinical evidence of **acute myocardial ischaemia** with
- Detection of **rise and/or fall of cTn values** with at least one value above the 99th percentile URL and
- At least one of the following:
 - Symptom of myocardial ischaemia
 - New ischaemic ECG changes (for type 1, 2, 3, 4a MI only)
 - Development of pathological Q waves
 - Imaging evidence of new loss of viable myocardium or new RWMA in a pattern consistent with an ischaemic etiology.
 - Identification of a coronary thrombus by CAG or autopsy (type 1)
 - Angiographic findings consistent with a procedural flow-limiting complication such as coronary dissection, occlusion of a major epicardial artery or a side branch occlusion/thrombus, disruption of collateral flow, or distal embolization. (type 4a)

Q. Clinical classification of MI – (Griffin) (ESC 2018 Guidelines – 4th universal definition)

- **Type 1 – Spontaneous MI** related to ischemia from a **coronary plaque rupture, ulceration, fissuring, erosion or dissection** resulting in coronary thrombosis (most common)
- **Type 2 – (Supply/demand mismatch MI)** MI due to ischemia resulting from increased oxygen demand or decreased supply
- **Type 3 – (Suspected MI-related death)** Sudden cardiac death with symptoms of ischemia, new ST elevation, or LBBB or coronary thrombus
- **Type 4a – (PCI-related MI)** MI associated with PCI (within 48 hours)
- **Type 4b – (Stent/scaffold thrombosis)** MI associated with stent thrombosis
Acute (0-24h), **subacute** (>24h-30d), **late** (>30d-1yr), **very late** (>1 yr after stenting)
- **Type 4c – (Restenosis associated with PCI)**
- **Type 5 – (CABG-related MI)** MI associated with CABG (within 48 hours)

Coronary procedure-related MI ≤ 48 hours after the index procedure is arbitrarily defined by an elevation of cTn values **> 5 times** for **type 4a MI** and **> 10 times** for **type 5 MI** of the 99th percentile URL in patients with normal baseline values.

Q. Classify MI – according to time of presentation after the onset of chest pain.

- **Hyperacute MI** – within 1 hour (hours) (broad-based tall T waves)
- **Acute MI** – upto 7 days (1 week)
- **Recent MI** – 7-28 days (1 month)
- **Old MI** – after 28 days (Older than 1 month)
- **Reinfarction** – within 28 days
- **Recurrent MI** – after 28 days

Q. Haemodynamic subsets (4) of AMI –

(based on CO & pulmonary oedema)

I – Normotension + clear lungs > requires no Rx for HF
(eg. Inf MI without RVI)

II – Normotension + bilatereal lung creps > due to moderate LV dysfunction. Rx with vasodilators & diuretics

III – Hypotension + clear lungs > due to Inf MI with RVI or concomitant hypovolaemia. Give fluid challenge & consider PAWP to guide therapy

IV – Hypotension + bilateral lung creps > Extensive MI & poor prognosis. Consider IABP, vasodilators, diuretics, inotropes.

Q. Diagnostic criteria of acute rheumatic fever – Revised Jones Criteria for ARF (Mir Jamal sir)

A. For all patients populations with evidence of preceding GAS infection –

Diagnosis: initial ARF	2 Major manifestations or 1 major plus 2 minor manifestations
Diagnosis: recurrent ARF	2 Major or 1 major plus 2 minor or 3 minor

B. Major criteria –

Low-risk populations	Moderate- to high-risk populations
Carditis – • Clinical and/or • Subclinical (echocardiographic valvulitis)	Carditis – clinical and/or subclinical
Arthritis – Polyarthritis only	Arthritis – • Monoarthritis or Polyarthritis • Polyarthralgia
Chorea	Chorea
Erythema marginatum	Erythema marginatum
Subcutaneous nodules	Subcutaneous nodules

C. Minor criteria –

Low-risk populations	Moderate- to high-risk populations
Polyarthralgia	Monoarthralgia

Fever ($\geq 38.5^{\circ}\text{C}$)	Fever ($\geq 38^{\circ}\text{C}$)
ESR ≥ 60 mm in the first hour and/or CRP ≥ 3.0 mg/dL	ESR ≥ 30 mm in the first hour and/or CRP ≥ 3.0 mg/dL
Prolonged PR interval, after accounting for age variability	Prolonged PR interval, after accounting for age variability

Low-risk populations are those with ARF incidences ≤ 2 per 100000 school-aged children or all-age RHD prevalence of ≤ 1 per 1000 population per year.

Q. Carditis of acute rheumatic fever - (Wadud sir)

Pancarditis.

- **Pericarditis** – chest pain, fever; pericardial rub & pericardial effusion (tachycardia – SA node is situated sub-epicardially)
- **Myocarditis** –
 - Inappropriate tachycardia (জ্বরের তুলনায় অনেক বেশী, অথবা জ্বর নাই),
 - Regurgitant murmur (MR) due to ring dilatation
 - Pulsus alternans due to severe LV dysfunction (Severe myocarditis)
- **Endocarditis** – valvulitis (Carey-Coomb murmur - MDM)
(Conduction defects – AV node is situated sub-endocardially)
- Causes of tachycardia: Pericarditis (SA node), Myocarditis

Q. Secondary prophylaxis in acute rheumatic fever - (Wadud sir) (Griffin 5e, p.303)

Disease status	Duration of therapy
RF + carditis + residual VHD	At least 10y post-episode and at least until the age of 25y; lifelong prophylaxis may be required for more severe VHD
RF + carditis, without VHD	10y or at least till 25y, whichever is longer
RF without carditis	5y or at least till 18y, whichever is longer
RF after valve surgery	Lifelong

- **Benzathine penicillin (Penicillin G):**
 - 1.2 million U ($>27\text{Kg}$), 0.6 million U ($\leq 27\text{Kg}$)
 - IM, every 4wk (every 3 wk for high-risk individuals or living in endemic areas)
- **Penicillin V:** 250 mg, 12 hrly
- **Sulfadiazine:** 500 mg daily ($\leq 27\text{Kg}$), 1g daily ($>27\text{Kg}$)
- **Erythromycin:** 250 mg 12 hrly

Q. T-wave alternans (Repolarization alternans) – (Griffin 5e, p.68)

- Higher sensitivity and specificity for inducible ventricular arrhythmias during EP testing.

Q. Causes of diastolic murmur at apex – (Gaffar sir)

- True MS
- Carey-Coomb's murmur (Due to valvulitis/ endocarditis in ARF)
- Austin-Flint murmur
- Large PDA, Large VSD
- Severe MR
- Prolapsing LA myxoma
- Ball-valve thrombus

Q. Murmur of MS vs Carey-Coomb's murmur – (Gaffar sir)

Murmur of MS	Carey-Coomb's murmur
S1 – loud	S1 – soft
Presystolic accentuation, if sinus rhythm	No presystolic accentuation
Opening snap	No opening snap

Q. Arthritis of ARF vs Post-streptococcal reactive arthritis – (Gaffar sir)

Arthritis of ARF	Streptococcal reactive arthritis
Incubation period: 2-3 weeks	Short incubation period
Migratory Polyarthritis	Not migratory
Characteristic response to aspirin	Not such
Frequent cardiac involvement, Renal involvement – rare	Frequent renal involvement, No cardiac involvement.

Q. Clicks - (Gaffar sir) (Griffin 5e, p.239)

- **Ejection click (Systolic clicks):** AS, PS (earlier in systole than the click of MVP)
- **Mid-systolic click:** MVP (at least 0.14s after S1), Septal & free wall aneurysm
- Vascular clicks: Root dilatation AR, TOF

Q. Postural drop - (Lutfor sir)

Q. Post-surgical scar in left chest - (Khaleq sir)

- Anterolateral thoracotomy:
 - o CMC
 - o Pericardiopleural window (done for recurrent pericardial effusion)
- Posterolateral thoracotomy:
 - o PDA corrective surgery
 - o CoA surgery
 - o Pneumonectomy

Q. Provocative tests for coronary spasm - (Khaleq sir)

- Hyperventilation
- Intracoronary acetylcholine
- Intracoronary ergonovine

Q. Unusual sites of ischaemic pain - (Khaleq sir)

- Occiput to sacrum

Q. Bifemoral approach in PCI - (Khaleq sir)

- CTO, left femoral – retrograde filling

Q. What are the PCI - (Khaleq sir)

- PTCA/ POBA
- PTCA with stent
- Rotational atherectomy
- Thrombectomy
- Angiojet

Q. ECG class- (Khaleq sir)

- MVP: T inversion in inferior leads (related to papillary muscle)
- Action potential of myocardial cell:
 - Phase 0 – QRS complex (R wave) [depolarization]
 - Phase 1 – J point [repolarization]
 - Phase 2 – ST segment [repolarization]
 - Phase 3 – T wave [repolarization]
 - Phase 4 – TQ segment [resting period of the cell]
- **Electrical systole and diastole:**
 - **Electrical systole:** QT interval (depolarization () & repolarization)
 - **Electrical diastole:** TQ segment
- LAE:
 - **Morris index:** $>0.04\text{mm-s}$ (depth $>1\text{mm}$ x duration $>0.04\text{s}$) of terminal negative portion of biphasic p wave in V1
 - P wave duration $>0.10\text{ sec}$ ($>2.5\text{mm}$)
- **Causes of LAE:**
 - MR (($\uparrow$ LAE than in MS),
 - MS,
 - AS,
 - HTN,
 - HCM,
 - PDA,
 - VSD
- **Causes of RAE:**
 - TS,
 - PS,
 - PH,
 - Cor pulmonale,
 - Ebsteins's anomaly
- **Causes of BAE:**
 - MVD with PH,
 - Cardiomyopathy,
 - Large ASD/PDA,
 - TV disease
- **Causes of RVH:**
 - PH
 - PS
 - Cor pulmonale
 - TOF
 - PAPVC
 - TR
 - RV type of HCM
- **Biventricular hypertrophy:** ECG criteria
 - LVH + one of the followings
 - Large equiphasic QRS complex (R+S) in V3-4 $\geq 60\text{mm}$ in children and $\geq 50\text{mm}$ in others (**Katz-Wachtel phenomenon**), commonly occurs in congenital heart disease (VSD or PDA with PH)

- RAD
 - Tall R waves in both right & left precordial leads especially R/S in V1 >1
 - Deep S waves in left precordial leads
- LAE + one of the followings
 - R/S in V5 or V6 ≤ 1
 - S wave in V5 or V6 $\geq 7\text{mm}$
 - RAD
- **Causes of biventricular hypertrophy:**
 - VSD,
 - PDA with PH,
 - AV canal defects,
 - MVD with PH,
 - HCM
- **Ventricular activation time (VAT):** The impulse takes to traverse the myocardium from endocardium to epicardial surface and is measured from the beginning of the Q wave to the peak of R wave.
 - Normally it is <0.03sec in V1-2 leads and <0.05sec in V5-6 leads
- **Peaked T waves: Causes**
 - Early STEMI (De Winter's T waves),
 - Prinzmetal angina,
 - Hyperkalaemia,
 - True posterior MI
-